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OCCURRENCE AND AETIOLOGY OF LUNG CANCER IN NORWAY IN THE LIGHT OF PATHOLOGICAL ANATOMY*

BY

LEIV KREYBERG

From the Institute of General and Experimental Pathology, University of Oslo

Lung cancer is one of the most fascinating human cancer problems of to-day. That this form of cancer, in a series of countries, has increased to a terrifying degree in this century, is no longer doubted. A number of facts are, however, still unknown, or disputed. As the incidence of lung cancer varies considerably between the many countries which show a definite increase, and as conditions of life, social as well as personal, also vary, it is not unreasonable to expect to derive valuable information regarding the development of lung cancer from comparative geographic studies.

In the present paper an attempt is made to elucidate the lung cancer problem through an epidemiological study in a country where the rise in incidence is still moderate, and where lung cancer development may be observed nearly in its *status nascendi*.

Fig. 1 (kindly supplied by Dr. Pedersen of the Norwegian Cancer Registry) shows a very moderate rise in lung cancer mortality in females in town and country from 1929–31 to 1952–54. A slightly more marked rise is seen in males living in the country, but the males living in urban conditions, according to our official administrative designation, show a very different picture. Here, already in the 1930's, a definite rise can be observed, a rise which since the middle of the 1940's has become very marked.

Seven years ago I planned a systematic study of the lung cancer situation in Norway in close co-operation with the two largest surgical thoracic units in our

* Given as a "Special University Lecture" at the University of London on May 1, 1956.

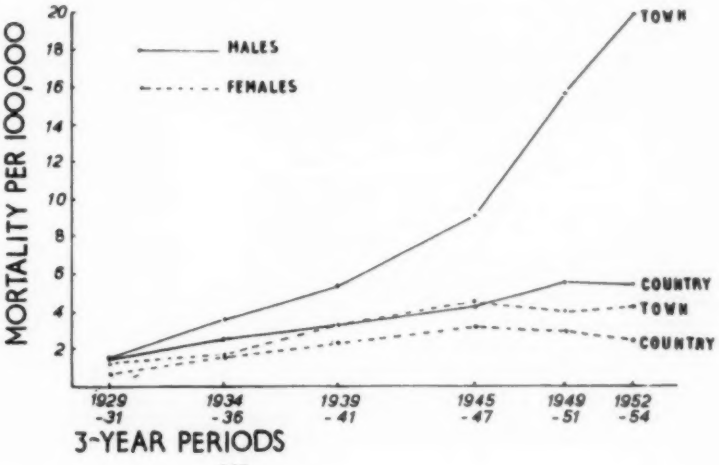


FIG. 1.—Mortality from lung cancer per 100,000 of the population in 3-year periods, by sex, in town and country, adjusted to standard population, 1950 (after Pedersen).

country: the Kirurgisk Avdeling A, Rikshospitalet (Prof. L. Efskind) and the III Avdeling, Ullevål Sykehus (Prof. C. Semb). From the beginning of the study it was evident that the general diagnosis of "lung cancer" includes a variety of tumours. The material for the present investigation has been limited to primary epithelial lung tumours.

Histology.—Considering the very different histological pictures, it would be strange if, within this limited group, different oncological entities were not represented. The first task, therefore, was to make a careful histological analysis of the material, and the next was to study the groups and sub-groups found as regards certain clear biological characteristics and possible differences, such as sex distribution and age. The results of this histological analysis of 300 primary epithelial lung tumours, mainly from surgical cases, are shown in Table I.

TABLE I

HISTOLOGICAL TYPES AND SEX DISTRIBUTION OF MATERIAL OF PRESENT STUDY

Group	Type of Tumour	Sex		
		Male	Female	Total
I	Squamous cell carcinomata	147	2	149
	Large cell carcinomata ..	12	0	12
	Small cell carcinomata ..	54	3	57
	Total	213	5	218
II	Adenocarcinomata ..	19	17	36
	Bronchiolar carcinomata	7	5	12
	Adenomata	13	12	25
	Salivary gland tumours ..	6	3	9
	Total	45	37	82
Total	.. ..	258	42	300

Sex.—The different incidence of these seven histological types in males and females is most striking. From the point of view of sex distribution the types of tumour fall into two distinct main groups: Group I, in which the male sex is predominant (213 : 5), and Group II, in which the two sexes are nearly evenly represented (45 : 37).

Age.—A study of age incidence reveals further marked differences (Figs 2 and 3). The curves in these Figures indicate "quotients" and not tumour incidence in the usual sense of this designation. A "quotient" is the number of different tumours in the whole material collected during the years 1948–1955, against the background of the size of the population in each age-group in a single year, *viz.*, 1950.

Fig. 2 shows the quotients for squamous cell carcinomata (Curve A) and for large cell and small cell carcinomata jointly (Curve B) in males only. These types of tumour correspond to Group I in Table I. The curves are nearly identical, and it seems permissible, in spite of certain histological differences, to conclude that these three types represent closely related tumour entities.

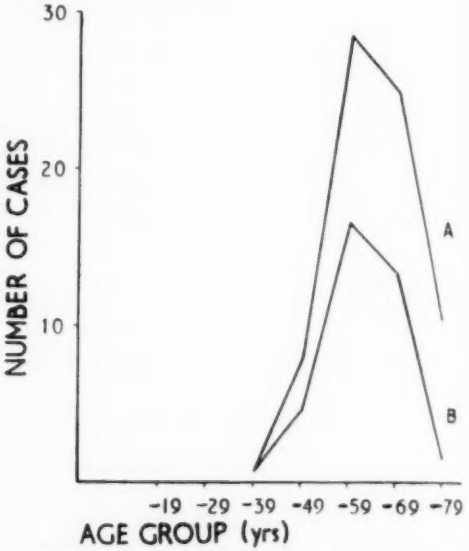


FIG. 2.—Relative frequency of (A) squamous cell, and (B) large and small cell carcinomata in males, by age groups.

In Fig. 3, Curve A represents adenocarcinomata, and Curve B adenomata and salivary gland tumours, including the benign, semi-malignant, and malignant varieties. These curves embrace male as well as female patients. Again, distinct and very different patterns of incidence are found, the adenocarcinomata steadily increasing with advancing years like

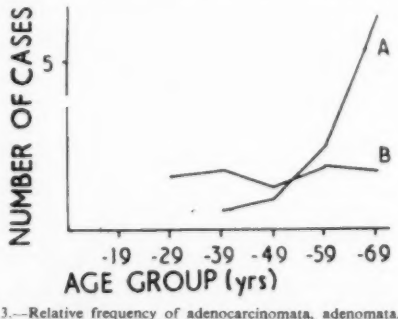


FIG. 3.—Relative frequency of adenocarcinomata, adenomata, and salivary gland tumours, in males and females, by age groups.
A = Adenocarcinomata.
B = Adenomata and Salivary Gland Tumours.

many other human tumours, and the adenomata and salivary gland tumours occurring with a uniform frequency in all adult age groups. Our own experience and reports in the literature show that the latter are not infrequent in very young persons, and may even occur in children. Our youngest patient is a boy of 12 years with a malignant adenoma who was reported upon by Harbitz (1937).

DIFFERENTIATION OF GROUP I AND GROUP II.—The main conclusions of this analysis are as follows:

(I) *Squamous cell carcinomata*, and *large and small cell carcinomata*, the types of lung tumour generally regarded as connected with special irritants, occur with a marked preponderance in males, and show a characteristic age curve. They very rarely occur before the late thirties. Until the last few years the maximum occurrence was in the fifth decade with a definite decline in the sixth, but recently a shift of this peak to the sixth decade has been observed expecially for squamous cell carcinomata. These observations indicate the existence of a new carcinogenic situation influencing the occurrence of the Group I tumours. In this connexion the analysis of the figures for England and Wales by Korteweg (1951) should be consulted. A similar development has also been recorded in Norway (Fig. 4).

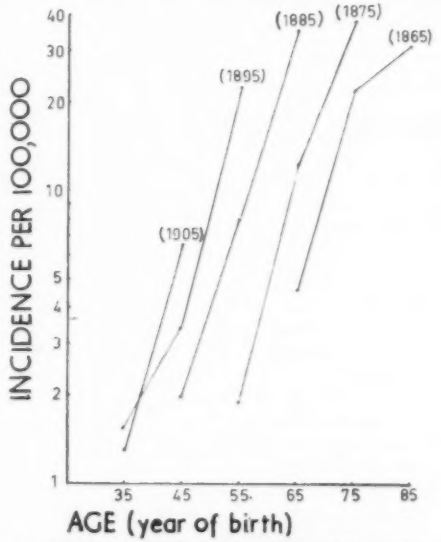


FIG. 4.—Mortality from lung cancer per 100,000 of the population, by age, showing year of birth (after Pedersen).

(II) *Adenocarcinomata*, according to our diagnostic criteria, embrace malignant tumours composed of more or less atypical, secreting or non-secreting, columnar or irregular polyhedral cells, with a more or less marked gland formation. These tumours, showing a nearly identical sex distribution and a steadily increasing frequency with advancing years, are most probably caused by weak carcinogenic agents, well-established in our society and acting with equal strength in both sexes.

Bronchiolar cell carcinomata form a small group and occur in our material in all adult age groups and in both sexes alike. They may be caused by unknown agents acting at random.

The histological picture, the equal sex incidence, and the lack of preference for any particular age group, together indicate that *lung adenomata* and *salivary gland tumours* are caused by accidental factors, presumably of developmental origin.

Lung cancer is accordingly histologically, as well as biologically, a heterogenous group, and this fact has been taken as the basis for the following analysis of the situation in Norway. The lung cancer material has been divided into the two main groups described above:

Group I tumours comprise squamous cell, large-cell, and small-cell carcinomata, with a wide sex difference, increasing numbers in recent years, and an accepted relationship to certain external irritants. Group II tumours comprise adenocarcinomata, bronchiolar cell carcinomata, adenomata, and salivary gland tumours, with an equal sex incidence and no connexion with any known irritants.

CANCER INCIDENCE IN EARLIER PERIODS.—In two laboratories (Rikshospital and Oslo City Hospital, Ullevål) two *post-mortem* series of cases of lung cancer have also been examined, one by Christiansen (1953) and the other by Jakobsen (1953). Table II shows the histological composition of this cadaver material compared with that of the present, mainly surgical, material.

TABLE II

HISTOLOGICAL TYPES AND SEX DISTRIBUTION OF TUMOUR MATERIAL FROM THREE PREVIOUS STUDIES COMPARED WITH PRESENT SERIES

Group		I		II	
Sex		Male	Female	Male	Female
Previous Series	Christiansen (1925–44)	25	11	15	13
	Jacobsen (1937–46)	46	8	24	22
	Christiansen (1945–52)	40	4	14	12
Present Series	Kreyberg (1948–55)	213	5	45	37

The equal occurrence of Group II tumours in the two sexes in each period is in strong contrast to the gradual but marked changes in the incidence of

Group I tumours, which are especially noticeable from the 1940s onwards.

Table III shows the ratio of Group I to Group II tumours in males in the cadaver and surgical series.

TABLE III

RATIO OF GROUP I TUMOURS TO GROUP II TUMOURS IN MALES IN SAME SERIES AS TABLE II

Author	Period of Study	Group		Ratio I : II
		I	II	
Christiansen ..	(1925–44)	25	15	1·7 : 1
Jakoben	(1937–46)	46	24	1·9 : 1
Christiansen ..	(1945–52)	40	14	2·9 : 1
Kreyberg ..	(1948–55)	213	45	4·7 : 1

DOMICILE.—The incidence of tumours of Groups I and II from the present series in the different counties of Norway is shown in Table IV. If the number of Group II cases is set against the background of the total population in each county, the frequency is seen to be of the same order of magnitude in Oslo, the capital, where the best diagnostic facilities are available, as in the remote counties in the far north. These figures further strengthen the previous indications that the Group II tumours are independent of regional or social conditions. With our present knowledge, they are the basic, and so far unavoidable, tumours of unknown aetiology.

TABLE IV

DISTRIBUTION OF HISTOLOGICAL GROUPS BY COUNTIES

County of Domicile	Group I Tumours			Group II Tumours			Total
	Male	Female	Total	Male	Female	Total	Population
Oslo* ..	77	2	79	6	10	16	286,222
Ostfold* ..	16	—	16	4	—	4	178,449
Akershus ..	9	—	9	5	3	8	301,149
Hedmark ..	4	1	5	6	—	6	169,525
Opland ..	1	1	2	2	2	4	154,734
Buskerud ..	8	—	8	3	2	5	149,948
Vestfold ..	14	—	14	3	1	4	147,555
Telemark ..	12	—	12	2	—	2	131,679
Aust-Agder ..	3	—	3	2	2	4	74,861
Vest-Agder ..	9	—	9	1	3	4	93,980
Rogaland ..	9	—	9	1	—	1	202,252
Bergen ..	8	—	8	—	—	—	110,424
Hordaland ..	3	—	3	1	1	2	188,389
Sogn-Fjordane	1	—	1	—	2	2	96,849
More-Romsdal	8	—	8	2	1	3	182,859
S. Trondelag (including Trondheim) ..	9	—	9	2	1	3	193,912
N. Trondelag	1	1	2	1	—	1	105,679
Nordland ..	6	—	6	—	5	5	215,972
Troms ..	4	—	4	1	—	1	113 722
Finmark ..	5	—	5	1	3	4	58,790
Sailors ..	2	—	2	1	—	1	—
Unknown ..	4	—	4	1	1	2	—
Total ..	213	5	218	45	37	82	

* Part of greater Oslo, including important industrial areas, was ormerly situated in Akershus and the figures presented in this Table refer to conditions before the inclusion of these areas in Oslo.

The Group II tumours in our material are thus fairly representative for the whole country. If this holds good for the Group II tumours, then the figures for the Group I tumours should also be representative, because all lung tumour patients show very similar symptoms, have passed through the same diagnostic procedures, and have been given the same diagnostic opportunities. The histological diagnosis separating the two groups is a late link in the diagnostic chain, often not obtained before the patients have arrived at the special hospital.

The incidence of Group I tumours is remarkably different in different counties; they are most numerous in Oslo, and next, through to a lesser degree, in those counties with the most urban and industrial areas.

Each patient was examined with respect to domicile from decade to decade, and the population was found to be remarkably stable until recently, when the flow from the countryside to the towns and industrial centres has increased.

Types of domicile are shown in Table V, Section A (opposite):*

(*i*) Countryside.
(*ii*) Smaller non-industrial towns.
(*iii*) Industrial centres.
(*iv*) Larger towns.

The Group II tumours are evenly distributed between the two sexes, regardless of domicile. The ratios of Group I to Group II tumours in males are lowest in the country and highest in the larger towns. The ratio in the country corresponds to the ratio in the early *post-mortem* series (Table III). The smaller non-industrial towns, mainly commercial and administration centres, show a somewhat higher ratio than the industrial centres, a result which at first I found puzzling.

AIR POLLUTION AND URBAN AND RURAL SURROUNDINGS.—These types of domicile were then examined as regards air pollution (Table V, Section B, opposite).

The countryside and smaller non-industrial towns were classified as having "fresh" air ([*i*] and [*ii*]), and the industrial centres and larger towns as having "polluted" air ([*iii*] and [*iv*]). The ratio in males is much higher for the latter than for the former.

When the same material is classified into "rural" or "urban" surroundings (Table V, Section C, opposite), the ratio in males is even greater.

These figures do not permit any decisive conclusions as to the influence of air pollution in the Norwegian material. In co-operation with Dr. J. M. Campbell of The Department of Pathology,

* This Table excludes sailors and patients of unknown domicile.

TABLE V

DISTRIBUTION OF HISTOLOGICAL GROUPS BY PATIENT'S DOMICILE

Group		I			II			Ratio of I : II in Males
Sex		Male	Female	Total	Male	Female	Total	
(A) Type of Domicile	(i) Country	31	2	33	17	17	34	1·8 : 1
	(ii) Non-Industrial Towns	45	1	46	8	5	13	5·6 : 1
	(iii) Industrial Centres	26	0	26	6	3	9	4·3 : 1
	(iv) Larger Towns*	87	2	89	7	11	18	12·4 : 1
(B) Air	Fresh (i, ii)	76	3	79	25	22	47	3·0 : 1
	Polluted (iii, iv)	113	2	115	13	14	27	8·7 : 1
(C) Surroundings	Rural (i)	31	2	33	17	17	34	1·8 : 1
	Urban (ii, iii, iv)	158	3	161	21	19	40	7·5 : 1

* Oslo, Bergen, Trondheim.

St. Bartholomew's Hospital, London, a study of the quality and the quantity of suspended matters polluting the air was carried out in Oslo and a few other Norwegian localities (to be published). The qualitative composition of the dust and soot retained on a filter paper after forced passage of air, is very like that found in English towns and in the English countryside (Fig. 5).

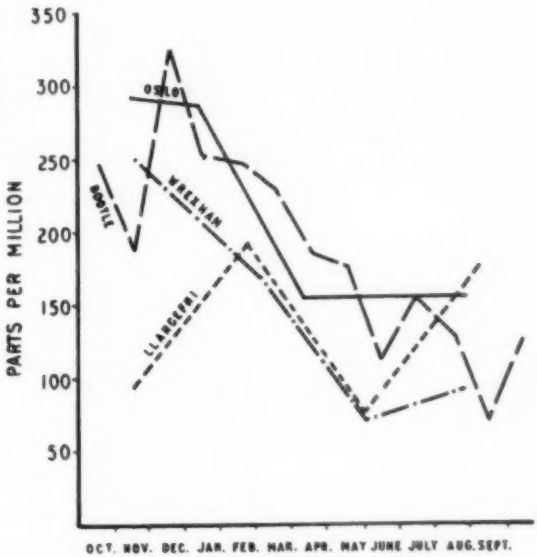
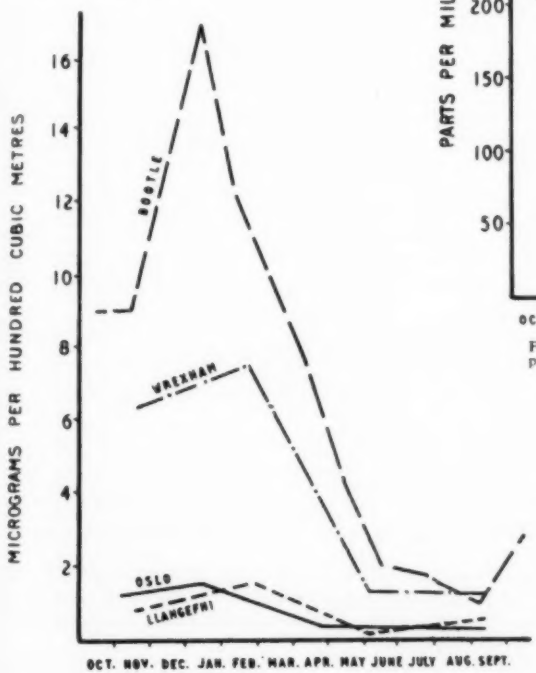


FIG. 5.—Monthly concentration of 3 : 4 benzpyrene as a proportion of the suspended impurities in the atmosphere in Oslo, Bootle, Llangefni, and Wrexham.



Quantitatively, it is found that the degree of pollution is very much lower, the figures for Oslo, the largest town in Norway, being only of the same order of magnitude as those in Llangefni, a small coastal village in Anglesey, North Wales (Fig. 6).

FIG. 6.—Monthly concentration of 3 : 4 benzpyrene in the air in Oslo, Bootle, Llangefni, and Wrexham.

A direct comparison of the lung cancer situation in Great Britain and Norway involves many known and unknown fallacies. Stocks and Campbell (1955), and all the earlier writers, treat "lung cancer" as an entity, as do our official mortality statistics. In both countries lung cancer mortality in rural areas has shown a negligible increase in the last 20 to 30 years, in a dramatic contrast to that in the towns. The present Norwegian analysis shows that this urban increase has been caused by an increase in Group I tumours only. Now, if air pollution could be accepted as an important causative factor for lung cancer in Great Britain, it would be difficult to accept such a conclusion as regards conditions in Norway. The very marked, nearly explosive, rise in the frequency of Group I tumours in males in Oslo, especially in the last 10 years, cannot be explained by a corresponding increase in air pollution in Oslo, beginning some 20–40 years ago (which is the time factor to be used for such calculations) and finally resulting in a pollution no greater than that found in a coastal village in Wales. In addition, no increase in lung cancer in females has been observed in Oslo in the same period.

OCCUPATION.—The incidence of lung tumours has also been examined in relation to the occupations of the patients (Table VI).

In most cases categorization was simple because the patients had followed a consistent line in their occupational life. Where the patient had changed his occupation, an estimate was made of the individual situation, and he was listed under the more specialized occupation if such work had lasted for 10 years or more. If, for instance, a ship's stoker had worked as such for 10 years or more and later came ashore and settled as a fisherman, he would be listed as a stoker. If, on the other hand, a former sailor had come ashore and worked in a copper mine for 10 years or more, he would be listed as a miner. Such doubtful cases were rare enough not to invalidate the general conclusions. This mode of approach was chosen in the hope of discovering any marked accumulations of lung tumour cases in any special occupation.

Table VI again indicates the even distribution of Group II tumours in the population, the largest numbers occurring in the occupations with the largest number of men. The Group I tumours show a more irregular distribution, the low numbers in farmers being most conspicuous. No special occupation is overwhelmingly represented, except perhaps workers in mechanical workshops, including solderers, welders, etc., but the total number is too small for accurate analysis. It may also be noticed that cases in civil engineers are comparatively numerous.

In order to assess the lung cancer risks on a

TABLE VI

DISTRIBUTION OF HISTOLOGICAL GROUPS BY PATIENT'S OCCUPATION AND SEX

	Occupation	I		II	
		Male	Female	Male	Female
(a) Open Air and House Work	Sailors	18	—	5	—
	Fishermen	6	—	—	—
	Farmers	9	—	12	2
	Timbermen, carpenters	7	—	—	—
	Gardeners, park attendants	3	—	1	—
	Builders	7	—	1	—
	Horse drivers	2	—	—	—
	Conductors (street car, rail)	3	—	—	—
	Patrol-policemen	1	—	2	—
	Telephone line workers	1	—	—	—
	Dockers	3	—	—	—
	Ships cooks	2	—	—	—
	House workers	—	4	—	28
	Total	62	4	21	30
(b) Clerical and Professional Work	Office clerks	14	1	5	3
	Business men and bankers	15	—	3	3
	Commercial travellers	3	—	—	—
	Civil engineers	10	—	—	—
	Lawyers	2	—	—	—
	Physicians	2	—	—	—
	Dentists	1	—	—	—
	Authors	1	—	—	—
	Singers	1	—	—	—
	Hotel owners, caterers	2	—	—	—
	Storehouse superintendents	1	—	—	—
	Telegraph operators	1	—	—	—
	Students	—	—	1	—
	Total	53	1	9	6
(c) Dusty Work	Firemen, stokers (land, sea)	5	—	1	—
	Engineers (land, sea)	5	—	—	—
	Locomotive drivers	1	—	—	—
	Chauffeurs	4	—	1	—
	Mechanical workers, grinders, solderers	26	—	5	—
	Blacksmiths	1	—	—	—
	Plumbers	6	—	—	—
	Miners (iron, copper, etc.)	5	—	—	—
	Goldsmiths	1	—	—	—
	Stone and concrete workers	5	—	—	—
	Masons	3	—	—	—
	Brick workers	1	—	—	—
	Glass workers	1	—	—	—
	Match workers	1	—	—	—
	Painters	5	—	—	—
	Vulcanizers	—	—	1	—
	Dyers, chemical workers	1	—	1	—
	Printers	1	—	—	—
	Shoe makers	4	—	—	—
	Tailors	3	—	—	—
	Furniture makers	4	—	2	—
	Cork workers	—	—	1	—
	Paper workers	2	—	—	1
	Textile workers	—	—	1	—
	Tar workers	—	—	1	—
	Bakers	3	—	—	—
	Butchers	3	—	1	—
	Brewers	1	—	—	—
	Fishcanners	1	—	—	—
	Warehousemen	2	—	—	—
	Waiters	3	—	—	—
	Total	98	0	15	1

broader basis, the different occupations have been grouped in three main categories;

(*a*) "Open air" and "house" work,
(*b*) "Clerical" and "professional" work,
(*c*) "Dusty" work.

When these categories had been decided upon and delimited, the Statistisk Sentralbyrå was kind enough to furnish a fairly accurate estimate of their numbers in the total population.

The analysis in Table VI was made in order to find whether certain occupations were especially liable to be connected with the development of lung tumours. The figures from the Statistisk Sentralbyrå give the situation at one particular time, and our patients were re-grouped accordingly so that each was listed under the occupation in which he was actually engaged at the time of diagnosis. This resulted in the following changes:

Sixteen men of 70 years old or more who had retired had previously been listed under their previous occupations, and thirteen men had changed their occupation, though several of these remained in the same main category. When the final corrections were made, the figure for "open air" and "house" work was reduced by four, with a corresponding increase of two in each of the other two categories. There was no change in the occupations of the women.

As the female cases of lung cancer in Norway nearly all manifest Group II tumours, it is of great interest, as a control of the figures received from the Statistisk Sentralbyrå, to examine the incidence of these tumours by sex and occupation (Table VII). Considering the restricted number of cases, a remarkable correspondence is found between the number to be expected if no special factors were involved, and the number observed. Our present material included only five cases of Group I tumours in females; the incidence of these tumours also is such as would be expected if no occupational hazards were involved.

The figures for males, however, show a very different picture. Group II tumours occur, as in the females, in proportion to the size of each main occupational category. If the frequency in "open air" and "house" workers is set as index 1·0, the corresponding indices for the two other categories are 0·8 and 1·0 respectively.

Group I tumours are much less frequent among male "open air" and "house" workers than in the two other main occupational categories, and if similar indices of frequency are calculated, an index of 1·0 for the first category corresponds to indices of 1·8 and 2·3 respectively for the other two categories.

In the same material the ratios of Group I tumours to Group II tumours in males are 2·8 : 1, 6·1 : 1, and 6·7 : 1 (Table VIII).

TABLE VIII

RATIO OF GROUP I TUMOURS TO GROUP II TUMOURS BY OCCUPATION, MALES

Occupation	Group		Ratio I : II
	I	II	
"Open air" and "House"	58	21	2·8 : 1
"Clerical" and "Professional" ..	55	9	6·1 : 1
"Dusty"	100	15	6·7 : 1

The two different materials for control (*viz.* the general population and the lung tumour groups *inter se*) give nearly identical results, and this greatly strengthens the assumption that the differences found are real.

These findings neither prove nor exclude the possibility that occupational "dust" *per se* is an

TABLE VII

DISTRIBUTION OF HISTOLOGICAL GROUPS BY MAIN OCCUPATIONAL CATEGORIES AND SEX, AS PERCENTAGE OF TOTAL POPULATION IN EACH TYPE OF WORK

Sex	Occupation	Total Population		Group			
		Number	(Per cent.)	I		II	
				No.	Per cent.	No.	Per cent.
Female	"Open air" and "House"	854,005	77·8	4	80·0	30	81·1
	"Clerical" and "Professional"	173,406	15·8	1	20·0	6	16·2
	"Dusty"	70,869	6·4	0	—	1	2·7
	Total	1,098,280	100	5	100	37	100
Male	"Open air" and "House"	467,494	44·3	58 (1)*	27·2	21 (1)*	46·7
	"Clerical" and "Professional"	244,163	23·3	55 (1·8)	25·8	9 (0·8)	20
	"Dusty"	341,634	32·4	100 (2·3)	47·0	15 (1)	33·3
	Total	1,053,291	100	213	100	45	100

* Index of frequency for males given in brackets.

important factor in augmenting the chance of developing Group I lung cancers, but two observations tend to reduce the probability of such an assumption: first, the considerably increased risk which is also found in recent years among "clerical" and "professional" workers not exposed to such dusts, and secondly, the wide range of "dusty" occupations involved and the few victims within each special occupation. Our findings are so far in accord with those of Doll (1953) and Lickint (1953).

Considering the fact that in Norway the increase in Group I lung tumours in males is intimately connected with the urban mode of life and has no demonstrable connexion with a general air pollution, as well as the fact that males doing "clerical" and "professional" work are affiicted nearly as often as workers in "dusty" surroundings, it seems reasonable to suppose that the new carcinogenic situation is linked to the personal life habits of men, rather than to general exposure to smoke, fumes, and dusts from streets, motor vehicles, urban air, and industrial undertakings.

Furthermore, this analysis also indicates that sufferers with Group II lung tumours represent the most suitable control material for the study of aetiological factors in the development of Group I tumours.

TOBACCO-SMOKING.—The tobacco-smoking factor has been carefully examined in this series of cases. A questionnaire was completed, giving present age, age when smoking was commenced and (where applicable) when it was ended, and amount of tobacco smoked. The last item was given as number of cigarettes or cigars a day, or as grammes smoked per week in the case of pipe-smoking and hand-rolling of cigarettes, a cigarette counting as 1 g., and a cigar as 5 g.

The first part of the analysis takes the form of a comparison of the smoking habits of the lung tumour patients with the control material presented by H. J. A. Kreyberg (1954). The lung tumour material was histologically typed, placed in Group I or Group II, and tabulated according to the smoking levels as used in the control study. Next, a calculation was made of the number of cases to be expected in the different smoking levels of the control material if as many individuals were represented as in the lung tumour material, age group (Table IX).

Group II Tumours

Table IX shows that the figures for females are nearly identical in the two series, indicating that tobacco-smoking is of no importance as an aetiological facor in the development of Group II tumours in females.

The figures for males are nearly identical and the same conclusions may be drawn.

Group I Tumours

Table IX shows that in males the picture is very different. There are significantly fewer non-smokers, and a higher number of heavy smokers; even for the other smoking levels the same tendency can be observed, with a systematic deviation towards the extremes.

These figures closely follow the pattern observed by many other workers, and the findings point to a close relationship between tobacco-smoking and the development of Group I tumours (often designated "epidermoid" tumours) in males.

The figures for Group I tumours in females are very small and no comment is necessary.

As the Group I and Group II tumours in males seem to behave so differently in relation to tobacco-smoking, it may be of some interest to compare the two groups in more detail. The pertinent figures for males are given in Table X.

TABLE X

SMOKING LEVELS OF GROUP I AND GROUP II TUMOUR PATIENTS (MALES)

Smoking Level (g.)		0	1–4	5–9	10–14	15–19	20–24	25–29	30–49	50 +	Total
Group	I	3	4	41	78	29	20	15	18	5	213
	II	3	5	14	12	5	1	4	0	1	45
Group I Tumours Related to Smoking		–	–	27	66	24	19	11	18	4	169

TABLE IX

TOBACCO-SMOKING AND INCIDENCE OF LUNG TUMOURS COMPOSED WITH GENERAL POPULATION

Sex	Group	I					II				
	Smoking Level (g.)	0	1–14	15–24	25 +	Total	0	1–14	15–24	25 +	Total
Male	Lung Tumours	3	123	49	38	213	3	31	6	5	45
	Controls (General Population)	28·2	128·6	41·9	14·6	213	6·7	26·1	9·4	2·8	45
Female	Lung Tumours	3	1	1	0	5	27	9	1	0	37
	Controls (General Population)	4·2	0·7	0·1	0	5	28·5	7·3	1·1	0·1	37

It has already been assumed that these two "groups" are probably representative of the real occurrence of the histological types for the whole country during the period of this study. We may, therefore, also assume that the relationship between the two groups is representative.

The non-smoking part of the male population is represented in Table X by three cases from each group, giving a ratio of 1 : 1.

If the Group II tumour patients are representative of the total male population as regards tobacco-smoking, and if the Group II tumours have no relationship to tobacco-smoking, it may be assumed that the remaining smokers in the male population, like the non-smokers and in the absence of any association between smoking and lung-cancer, would produce Group I tumours in numbers corresponding to the Group II tumours at each smoking level.

If these assumptions are accepted, a simple deduction of the Group II tumour cases from the Group I tumour cases, smoking level for smoking level, will reveal the number of Group I tumours associated with tobacco-smoking (Table X). It seems that in Norway, to-day, approximately only one out of every five cases of Group I lung tumours in males has no association with tobacco-smoking. This proportion is considerably higher than that estimated for England and Wales, since the figure of 17 per cent. calculated by Doll (1953) for the age group 25 to 74 years also included the Group II cases and all the female cases, but the smallness of the absolute figures for non-smokers introduces a certain inexactness into the present study.

Table X also shows that in males smoking 4 g. tobacco a day or less—equivalent to four cigarettes—the incidence of the two tumour groups is very like that in non-smokers; they do not develop more Group I tumours, and thus no relationship with smoking has been established. As soon, however, as the amount of smoking is increased above 5 mg. per day, the ratio of Group I to Group II tumours increases steadily, and reaches the very impressive level of 23 : 1 when the limit of 30 g. a day has been passed (Table XI).

TABLE XI

RATIO OF GROUP I TO GROUP II TUMOURS BY SMOKING LEVELS (MALES)

Smoking Level (g.)	Group		Ratio I : II
	I	II	
0	3	3	1 : 1
1—4	4	5	0·8 : 1
5—9	41	14	2·9 : 1
10—19	107	17	6·3 : 1
20—29	58	6	9·7 : 1
30+	23	1	23·0 : 1

This does not only indicate a strong relationship between Group I lung carcinoma and smoking, but also shows the relative risks associated with different smoking levels. The figures are similar to those reported by Doll and Hill (1952), Levin (1954), Randig (1954), and Hamond and Horn (1955).

Whereas previous students of this problem have based their conclusions mainly upon the occurrence of all types of lung cancer, and have made their calculations on the basis of the population in general, the present study has utilized a totally different material for comparison. Here, the "control" material also consists of lung tumour patients, originally from the same sources as the material proper, and only separated from it by an histological analysis. This "control" material comes closer to the ideal material than any other. The present findings, therefore, strengthen considerably the conclusions of previous researchers. It would be strange, if another underlying primary factor should prove to be the real cause, and if the use of tobacco and the development of lung cancer should be two quantitively corresponding manifestations of such an unknown factor. At least, no such factor has been found, or even plausibly suggested.

The importance of the close association of four-fifths of the Group I tumours in males with tobacco-smoking should not, however, lead to neglect of other possible factors in the carcinogenic situation.

Tobacco-Smoking and Occupation.—A greater tendency to Group I lung cancer was observed in males with "dusty" occupations and trades, as well as in "clerical" and "professional" workers, as compared with men doing "open air" and "house" work, and it was emphasized that the dust, *per se*, might be of some importance.

A survey has been made of the smoking levels in males with Group I lung tumours according to their occupations (Table XII).* The material is, admittedly, small and accordingly difficult to analyse, but

TABLE XII

OCCUPATION OF MALES WITH GROUP I TUMOURS BY SMOKING LEVEL

Occupational Category	Smoking Level (g.)										Ratio I : II
	0	4	9	14	19	24	29	49	+	Total	
"Open Air" ..	3	2	13	24	5	6	5	2	2	62	2·8 : 1
"Clerical" and "Professional"			8	11	15	5	4	10	2	55	6·1 : 1
"Dusty"		2	20	43	9	9	6	6	1	96	6·7 : 1

* Sailors are included in Table XII.

there seems to be no indication that the comparatively higher incidence of Group I tumours in males in "dusty" occupations is associated with an especially high smoking level. The figures may rather be interpreted to mean that "dusty" occupations may add another carcinogenic insult to the effect of the smoking. Theoretically, then, besides the well-known lung carcinogenic agents, such as nickel, asbestos, coal tar, and uranium ore, certain "dusts" may also have an additive effect in the development of some cases of lung cancer, even if these agents are so weak as not to be easily or directly discovered. Exposure to metal dusts may especially be suspected. It is worth noting, however, that the 92 cases in "dusty" occupations include no non-smokers.

"Clerical" and "professional" workers among males with Group I tumours seem to be rather heavy smokers. Not one smoked less than 5 g., only eight out of 53 patients smoked less than 10 g. a day, and there were no non-smokers.

The "open air" and "house" workers represent the largest occupational category, but they show a low number of Group I tumour patients—only 62 cases in all. It seems that the smoking level is also generally lower than in the other occupational categories. This holds good not only for the relative number of smokers, but also for the amount smoked; all three non-smokers among the males with Group I tumours belong to this category.

Tobacco-Smoking and Domicile.—In this connexion it may be of interest to recall that, in the study of the geographical distribution of the Norwegian lung cancer material, it was observed that Group I tumours were more heavily represented in the "smaller non-industrial towns" with "fresh" air than in the "smaller industrial centres" with more or less "polluted" air. This finding was initially unexpected by the writer, through the predominant assumption that general air pollution has some importance for the development of lung cancer. The original paper comprised 235 cases of lung cancer, and figures of the present material, augmented to 300 cases, confirm the previous observation. The original "control" material gave no information as to smoking habits in different types of domicile, although the findings of more moderate smoking in rural districts was expected.

After the publication of the paper on geographical distribution, another material with a bearing on Norwegian smoking habits was placed at my disposal through the kindness of "Fakta", Oslo, an institute for marketing research, which had just completed a survey of one of our tobacco factories. Fig. 7 presents the proportions of smokers, as well

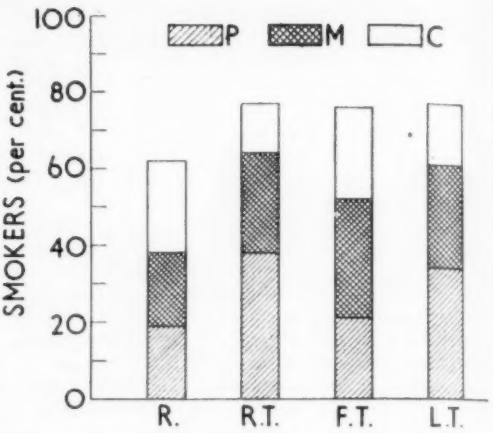


FIG. 7.—Smoking habits of males in different types of domicile (from "Fakta").

P = pipe-smokers.
M = mixed pipe- and cigarette-smokers.
C = cigarette-smokers.
R = rural districts.
T.R. = smaller non-industrial towns.
F.T. = smaller industrial centres.
L.T. = larger towns.

as the different types of smoking in areas corresponding to our "rural districts", "smaller non-industrial towns", "smaller industrial centres", and "larger towns". The figures presented are not comparable to the figures of our original "control" material, because of the different criteria used. In our controls as well as in our lung cancer patients, a smoker was defined as "a person who has smoked as much as 1 g. tobacco (in any form) daily for at least one year", whereas the "Fakta" material includes only current smokers. This explains the important differences in the percentages of smokers and non-smokers in the two series. The types of smoking in the different, geographical areas of the "Fakta" material are, however, comparable *inter se*, and Fig. 7 shows that smoking habits in the smaller towns differ markedly. The percentage of smokers is practically the same but the "smaller non-industrial towns", which are mainly commercial and administrative centres, show a considerably higher number of pure and mixed cigarette-smokers than the "smaller industrial centres", even if the average number of grammes smokes is the same.

The males with Group I tumours, in the present material also, show higher smoking levels in the smaller non-industrial towns than in the smaller industrial centres (Table XIII), opposite.*

* In Table XIII sailors have not been included.

TABLE XIII

DOMICILE OF MALES WITH GROUP I TUMOURS BY SMOKING LEVEL

Domicile	Smoking Level (g)										Ratio I : II
	0	4	9	14	19	24	29	49	+	Total	
Country-side ..	3		7	10	4	4	5			31	1·8 : 1
Non-Industrial Towns ..			10	16	3	3	4	8	1	45	5·6 : 1
Industrial Centres ..		2	3	11	5	2	1	2		26	4·3 : 1
Larger Towns ..		2	16	32	15	8	5	6	3	87	12·4 : 1

Types of Tobacco-Smoking.—Table XIV shows the types of smoking recorded for males with Group I and Group II tumours. There is a heavier representation of pure and mixed cigarette-smokers in Group I, and more pipe-smokers in Group II. The Group I series includes more smokers than the Norwegian "control" material presented by H. J. A. Kreyberg (1954). A detailed comparison with the "Fakta" material is useless, because of the different criteria used for non-smokers. The ratio of Group I to Group II tumours in pure pipe-smokers, pipe- and cigarette-smokers, and pure cigarette-smokers is shown in Table XV.

The absolute figures are small and the picture is not completely clear, so that no precise conclusions can be drawn. On the average, the pure pipe-smokers do not seem to use as much tobacco as the cigarette-smokers, and a quantitative factor may thus possibly also be of importance. It may, however, be worth calling attention to the apparent absence of increase in the ratio of Group I to Group II tumours in the pure pipe-smokers with the increasing amount of tobacco smoked, in contrast to the definite and systematic increase in the ratio when cigarette-smoking is involved. This may indicate that pure pipe-smoking does indeed carry a smaller risk of the development of lung cancer than cigarette-smoking.

TABLE XV

RATIO GROUP I TO GROUP II TUMOURS IN MALES BY TYPES OF SMOKING

Smoking Level (g.)	Type of Smoking			
	None	Pipe only	Pipe and Cigarette	Cigarette only
Under 10		2·3 : 1	4·0 : 1	1·9 : 1
10–19		4·0 : 1	9·7 : 1	6·4 : 1
20 + ...		1·0 : 1	16·0 : 1	13·3 : 1
Total	1 : 1	2·8 : 1	6·0 : 1	

Tobacco-Smoking and Sex.—A striking feature of the Tables is the different sex incidence of some types of lung cancer, even among non-smokers. As there are many more female non-smokers, and as many females who are only very moderate smokers, the absolute figures cannot be compared. But, if the ratio of Group I tumours to Group II tumours is again used in analysing male and female non-smokers (Table XVI), the result is a ratio of 1 : 1 in males (3 : 3 cases) and of 1 : 9 in females (3 : 27 cases).

TABLE XVI

RATIO OF GROUP I TUMOURS TO GROUP II TUMOURS BY SEA AND TOBACCO SMOKING

Sex	Tobacco-Smoking	Group		Ratio I : II
		I	II	
Male	Non-Smokers	3	3	1 : 1
	Smokers	210	42	5 : 1
Female	Non-Smokers ..	3	27	1 : 9
	Smokers	2	10	1 : 5

If the figures for smokers of less than 5 g. tobacco, in whom no carcinogenic association was observed in our material (Table XI), are also included, the same picture appears: a ratio of 1 : 1·1 in males (7 : 8 cases) and of 1 : 7·5 in females (4 : 30 cases).

With due allowance for the smallness of the numbers, this indicates that, even among non-smokers, males are predominant in Group I.

TABLE XIV

TYPES OF SMOKING IN GROUP I AND GROUP II TUMOUR PATIENTS AND SMOKING LEVELS

Group	Type of Smoking	Smoking Level (g.)									
		0	–4	5–9	10–14	15–19	20–24	25–29	30–49	50 +	Total
I	None	3	—	—	—	—	—	—	—	—	3
	Pure Pipe	—	3	11	19	1	2	—	—	—	36
	Pipe + Cigarette	—	—	12	18	11	4	5	3	4	57
	Pure Cigarette	—	1	18	41	17	14	10	15	1	117
II	None	3	—	—	—	—	—	—	—	—	3
	Pure Pipe	—	—	6	4	1	1	1	—	—	13
	Pipe + Cigarette	—	—	3	2	1	—	1	—	—	7
	Pure Cigarette	—	5	5	6	3	—	2	—	1	22

Wynder (1954), in his Table VI derived from his own material and from the literature, shows the occurrence of types of lung cancer (very much like our own grouping) in non-smoking males; this material gives a ratio of 2·4 : 1 (34 "Epidermoid" : 14 "Adenocarcinoma"), which is a very low ratio, even if not as low as the Norwegian one quoted above.

The fact that Wynder's collected material only concerns "Epidermoid" (including "oat-cell" carcinomata) and "Adenocarcinomata" (including bronchiolar cell carcinomata) partly explains the difference from the present material, in which the Group II tumours also include adenomata and salivary gland tumours. If the latter are omitted from the present material, we find a ratio of 1·8 : 1 for males (7 : 4 cases).

The ratio in females in our material is 0·1 : 1 (3 Group I : 27 Group II in non-smokers), and by Wynder's classification the ratio is 0·15 : 1 (3 Group I : 20 Group II).

These figures may appear to contradict the statement by Doll (1953) that "the incidence of lung cancer in non-smokers may be the same in men and in women and in residents in areas of different density of population", a statement with which Wynder agrees.

Possibly, however, these different statements may be reconciled. It should be emphasized that this marked sex difference in the Norwegian material applies to Group I tumours only. If the figures from Table XVI are used to compare all types of lung cancer seen as an entity—and that is the background of Doll's analysis—one finds a total of six "lung cancers" (3 + 3) in the non-smoking males and thirty (3 + 27) in the non-smoking females. As the non-smoking females in the population, however, are much more numerous than the non-smoking males, a factor of 5 is very probable according to our previously quoted figures, and one arrives at an approximately equal sex incidence of "lung cancer" among non-smokers.

As Group I tumours in the Norwegian material are rather uncommon among females as well as among non-smoking males, the peculiar sex difference in the incidence of the Group I tumours is easily obscured by the much greater number of Group II tumours, if special attention is not paid to the histological typing, followed by a separate treatment of the two groups. Thus the value of the full application of this analytical procedure has again been demonstrated.

This means that the possibility of a special sex disposition to the development of Group I tumours in males cannot be denied. Whether this disposition is based upon architectural differences in the gross anatomy of the lungs, upon different biological responses of the epithelium, or upon hormonal factors, is completely unknown; the question cannot yet be tackled by clinical and experimental methods, though gross anatomical studies might very well be made.

Summary and Conclusions

It has been shown that the increase in lung cancer in males which has taken place in Norway in the 20th century closely follows the pattern of development in a number of other countries, though delayed by a decade or two.

At first a very slow and parallel rise was seen in the number of cases of lung cancer reported in males and females, but gradually, and from the mid 1930's especially, the increase in male cases has greatly exceeded the increase in female cases. This sex difference is almost entirely confined to urban areas.

A study of the histological types of lung cancer has revealed that, from the time of the more pronounced increase in male cases, the relative frequency of the histological types also changed, with a steadily increasing number of male cases of Group I tumours (squamous cell, large cell, and small cell carcinomata). No such change was found in male cases of Group II tumours (adenomata, bronchiolar cell carcinomata, adenocarcinomata, or salivary gland tumours), or in females of either group. It is concluded that almost the whole of the reported increase in female cases, as well as part of the increase in male cases, is due to better diagnostic facilities, whereas the specific increase in Group I tumour cases in males is due to a real increase in the number of lung tumours. This means that a new carcinogenic situation manifested itself in Norway in the mid 1930s. Since these Group I tumour cases in males have a period of development ranging between 20 and 40 years or more, this new carcinogenic situation must have begun to arise at the beginning of the 20th century. It has been manifested, up to the present, in male patients in urban areas. From theoretical considerations, as well as from our special studies, it is thought unlikely to be due to general air pollution, although the new carcinogenic situation is mainly observed in urban areas. The main active principle(s) is (are) most probably connected with male working conditions and/or life habits.

It is well known that certain industrial processes dealing with radio-active material, nickel, asbestos, and coal tar are associated with an increased rate of lung cancer. The Norwegian material does not, however, show any special trade or occupation which carries a risk so marked that the increased

incidence of Group I tumour cases in males can be explained by occupational hazards.

Males in "dusty" occupations generally have an increased lung cancer frequency, as compared with "open air" workers, but the risk is nearly as great among "clerical" and "professional" workers.

The tobacco-smoking habits of 300 Group I and Group II lung cancer cases (213 males and 5 females in Group I, and 45 males and 37 females in Group II) have been investigated and compared with those of the general population, and the main conclusions are as follows:

(1) As no differences have been found, either in males or in females, between the smoking habits of the Group II tumour patients and the smoking habits of the population in general, it is concluded that tobacco-smoking has no relation to the occurrence of Group II lung tumours. Such tumours represent, in Norway today, nearly 90 per cent. of all female lung tumour cases, but less than 20 per cent. of all male cases.

(2) As considerably fewer non-smokers are found among the males with Group I lung tumours than among those with Group II tumours or than among the corresponding male "control" material, and as the ratio of Group I to Group II cases increases steadily with the amount of tobacco smoked, it is concluded that tobacco-smoking is closely related to the development of a considerable proportion of Group I lung tumour cases in males. The very limited female material does not contradict this conclusion.

(3) As a certain number of Group I tumours occur in male and female non-smokers, it is concluded that not all Group I tumour cases are related to, or influenced by, tobacco-smoking.

(4) From an analysis of the ratio of Group I to Group II tumours in males, it has been calculated that, in Norway at present, four out of five cases of Group I lung tumours in males are related to tobacco-smoking, and that one out of five arises from causes unrelated to tobacco-smoking.

(5) As males doing "dusty" work show a greater number of Group I lung tumours, in spite of more moderate tobacco consumption, than males doing "clerical" and "professional" work, it is tentatively suggested that industrial dusts and fumes may aggravate the injury caused by tobacco-smoking.

(6) A previously reported finding, that relatively more Group I tumour cases occur in males in smaller non-industrial towns (administrative and commercial centres) with "fresh" air than in smaller industrial centres with more or less "polluted" air, may be explained by the fact that the former type of community includes more cigarette-smokers.

(7) The relationship of lung cancer development to pipe-smoking, if present at all, is less marked than its relationship to cigarette-smoking.

(8) The ratio of Group I to Group II tumours in non-smokers is nine times greater in males than in females. This great difference *per se*, besides the fact that the only three males with Group I tumours in the present material who were non-smokers were among the "open air" workers, makes it difficult to ascribe the whole sex difference to external factors alone. A biological sex difference, architectural and/or biochemical, influencing the response of the lungs to some, or all, carcinogenic agents, cannot be excluded.

In the light of this discussion and of the conclusions presented, it may be of interest to emphasize the following points:

(1) The conclusions are based upon a special histological subdivision of "lung cancer" material, according to certain definite criteria. If the grouping and the criteria used are not followed, the same results can accordingly not be obtained. An off-hand dismissal of the possibility of making a sufficiently precise histological grouping is contradicted by the results. Not every case can be grouped with certainty, but most cases can.

(2) The results of the present study are so consistent and so clear, that it is unreasonable not to regard them as expressions of true conditions. An artefact with such a degree of consistency is most improbable.

(3) The figures quoted represent trends and relationships only. They do not express fixed mathematical correlations, generally applicable, but refer to conditions in Norway during the period of investigation. They cannot be applied to other countries and other conditions without further qualification. Differences in types of tobacco smoked, and in the amount and degree of smoking, differences in the surrounding air (in general or in workshops), and probably also constitutional differences, may cause minor quantitative variations in the effects, but the trends of these general conclusions I do regard as generally valid.

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COHORT ANALYSIS OF MORTALITY RATES AS AN HISTORICAL OR NARRATIVE TECHNIQUE

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The use of cohort analysis applied to mortality rates in human biology has been limited and obscured by pre-occupation with two concepts which fail to embrace the whole or probably even the larger part of the domain properly described by the method, though each concept is of legitimate interest in its own sphere. These two concepts are:

(1) That the shape of the mortality curve can be adequately described by a mathematical formula expressing a "law of mortality" [*e.g.* that of Gompertz (1825) or of Makeham (1860)].

(2) That if the mortality pattern either for all mortality or for a particular disease is fixed in some such manner, then the intensity with which the population is affected is determined by the early years of life of the members of that population. This early determination of the mortality pattern is referred to as the "generation" effect.

History

If cohort analysis is taken to mean the following of the development of some attribute, in this paper mortality rate, in a particular group of people defined by their birth date, the first unequivocal description of the method that I have been able to trace was that given by Andvord (1930). It related to the mortality from tuberculosis, and dealt with what has been defined as the "generation effect". An earlier paper by Derrick (1927) mentioning the "generation effect" for all mortality does not use cohort analysis, but a method that will be defined below as "age-contour analysis. A claim made in an editorial footnote in 1953 that Korteweg (1927) had first used the method in a study of mortality rates from tuberculosis is not borne out by reference to Korteweg's paper.

Kermack, McKendrick, and McKinlay (1934a, b) then take up the tale and emphasize the effect of mortality in the early years of life in determining subsequent mortality. They succeeded in disposing of the gloomy prophecy of Karl Pearson and his school (*e.g.* Pearson, 1912) that decreasing infantile mortality would inevitably increase mortality rates at higher ages because of a lack of proper natural selection.

About the same time Cramér and Wold (1934, 1935) had independently been studying the method from an actuarial standpoint, and had published two reports of a detailed cohort analysis of the mortality rates in Sweden. They explicitly referred to the work as a "study in graduating and forecasting". They came to the conclusion, when comparing the results of graduating both the cohort death rates and the "date-contour" death rates by the Makeham formula, the "neither graduation seems to be decidedly to be preferred to the other".

In a valedictory address to the Royal Statistical Society, Greenwood (1936) discussed the method in relation to "English Death Rates, Past, Present and Future", but seemed to be so obsessed by the actuarial demand for graduation and extrapolation that the narrative value of the method escaped him. He stated (p. 691) that ". . . the occurrence of epidemics, such as the cholera epidemics of the past or the influenza of our own time must prejudice arithmetically a diagonal [cohort] method more seriously than a columnar [date-contour] method". (Words in brackets inserted).

Barclay and Kermack (1937–38) extended cohort analysis to birth rates, and this started a development of technique that gave rise to studies, excellently described by Taylor (1952), that have changed our concept of fertility analysis but are not germane to the issues raised in the present paper.

In 1939 came the posthumous publication of work already verbally communicated in 1936 by Frost, where the term "cohort" was used and defined for the first time. This important paper deals with the shape of the mortality curve in tuberculosis and the variations in intensity of mortality rates from this

disease according to the date of birth. It was a forerunner of a series of papers on tuberculosis mortality which extended these observations to different countries but were concerned in the main with the "generation effect" (Picken, 1940; Moine and Oudet, 1947; Daw, 1950; Lancaster, 1950; Springett, 1950 and 1952; Spicer, 1954). The most comprehensive and penetrating discussion of cohort analysis that has so far appeared is to be found in the fundamental study reported by Springett (1950), and this paper should be consulted both for details of the way curves can change in general and the cohort analysis of tuberculosis mortality rates in particular.

Korteweg (1951) used cohort analysis to draw attention to the explanation of the apparently anomalous shape of the age-specific mortality curve for lung cancer, and this was followed by a series of papers (Korteweg, 1953; Levin, 1953; Dorn, 1953; Clemmesen, Nielsen, and Jensen, 1953; Cutler and Loveland, 1954) in which this artefact is referred to, though none of them makes it clear that this is a general effect of the rate of rise exceeding a critical value. Stocks (1953a, b) explored the cancer field more widely by also plotting the mortality curves for cancer of the uterus, female breast, and stomach but was concerned with the hypothesis of Nordling (1953) that cancer is the result of the seventh mutation of a cell, and that this can be deduced from cancer mortality rates. Haenszel and Shimkin (1956) attempted to use the narrative properties of the analytical system to determine whether the social history of smoking habits in the United States of America was compatible with the epidemiology of lung cancer as revealed by cohort analysis.

The Method

The principle underlying cohort analysis is so simple that an experimental biologist, who would unhesitatingly, if unwittingly, make use of the method in his animal experiments, might be surprised that any demographers feel that discussion of its logical credentials is necessary.

If we take a batch of new-born animals and wish to observe the mortality that occurs when they are kept under specified conditions, we record the number that die during successive periods of time and calculate the mortality rate from this number and from the number of survivors of the original batch. If we were to run a series of experiments, each batch being kept under different conditions, we should not attempt to make a mortality curve by taking mortality rates at different ages from different series of animals.

If our series of experiments is so large that we start each new one at a regular interval, this interval being the same as the intervals between calculating the mortality rates, we may enter the observations on a grid (Fig. 1) in which the top of each column represents the start of an experiment and the successive observations on any one batch of animals lie along a diagonal of the grid. If the grid record includes some experiments that had been started before the arbitrary zero date, all the cells of the grid can be filled with mortality rates.

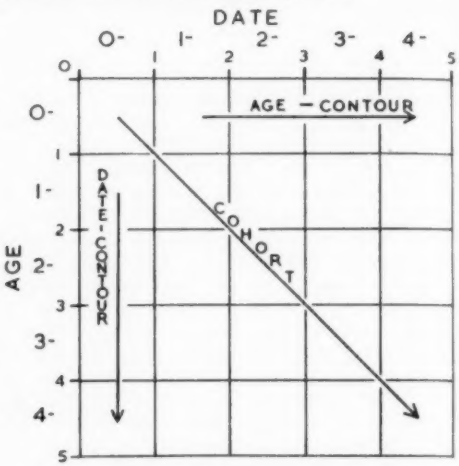

FIG. 1.—Principle of analysis by cohort and contour.

There are now three ways of traversing this grid:

(1) The diagonal, labelled "cohort", in which each successive square contains a mortality rate derived from the survivors of one experimental batch of animals.

(2) The vertical, labelled "date-contour", in which each successive square contains a mortality rate derived from a different batch of experimental animals, each batch kept under different conditions, although the ages defining each rate form a smooth progression.

(3) The horizontal, labelled "age-contour", in which each square contains a mortality rate derived from a different batch of animals, but in which the age of the animals referred to is the same.

Whilst it would not normally occur to us that the vertical series formed in any sense a biological description of the force of mortality characteristic of the type of animal used, since each rate is from a batch of animals kept by definition under different conditions, we might well use the horizontal series to see if the different experimental conditions had an effect on the mortality rate at any stated age. If, and

only if, no effect were found, and the rates along the horizontal line remained constant, the vertical line would become a valid description of the mortality pattern for all the batches of animals, for then the vertical lines and the diagonals would have become identical.

When analogous studies are made on human populations, the form of social accountancy that is perforce used tends to obscure this issue. In England and Wales, for example, the Registrar-General publishes an "Annual Report", and obviously he can only record the mortality that has occurred in that year, *i.e.* a series of rates lying along the "date-contour" of Fig. 1. Ideally it would seem that a grid constructed from mortality rates for each year of life, collected annually, would offer the best opportunity for cohort analysis, since the experience of the survivors of those born in each successive calendar year could be followed. In practice, it is not the custom to record mortality regularly at each year of age, but to divide the ages into a series of groups, often of 5 years.

When this is done, a somewhat complex situation occurs because the membership of a group of people who are aged between 50 and 55 in 1950 is not exactly the same as the membership of a group aged between 50 and 55 in 1951, even if no deaths have occurred.

This point is perhaps best shown by a graphical method, as in Fig. 2.

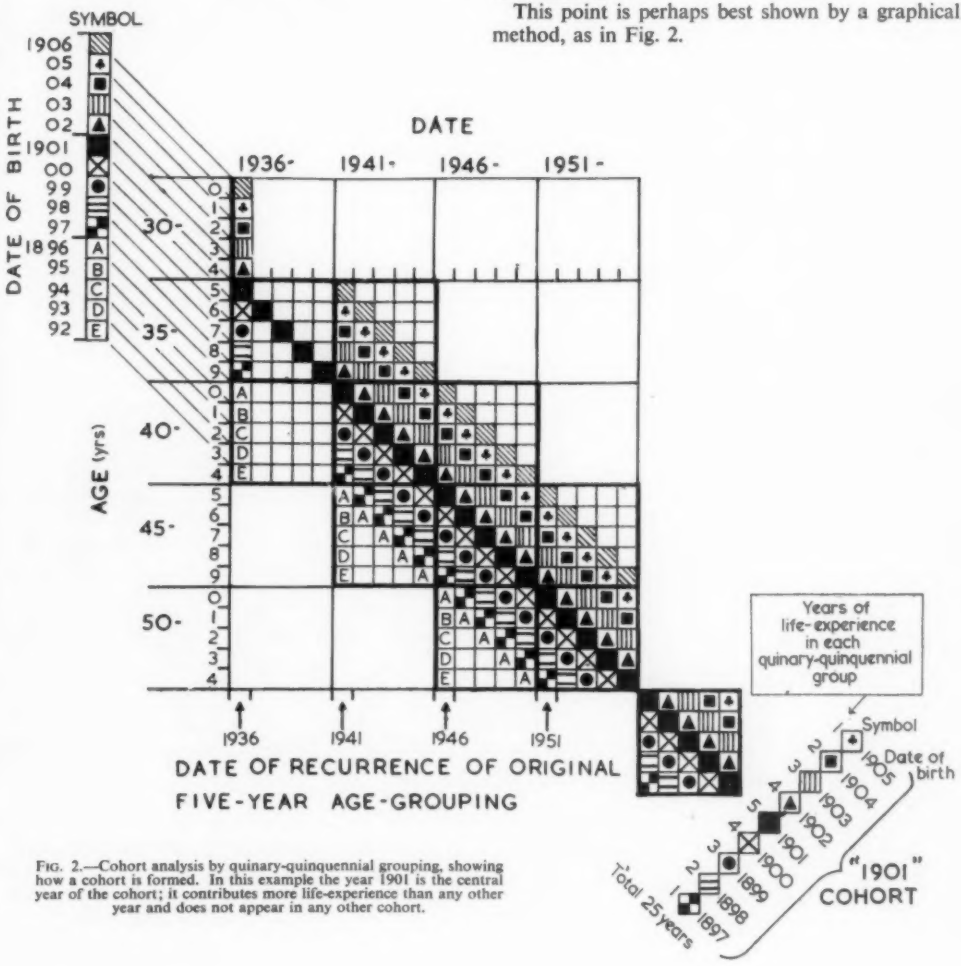

FIG. 2.—Cohort analysis by quinary-quinquennial grouping, showing how a cohort is formed. In this example the year 1901 is the central year of the cohort; it contributes more life-experience than any other year and does not appear in any other cohort.

If we allot a different birth symbol to each of the fifteen years 1892 to 1906 and then plot the movement of these symbols to the appropriate ages in a series of calendar years, we can see that birth-dates can be followed

(*a*) by following one year at yearly intervals,

(*b*) by taking any group of n consecutive birth-years and examining their experience every n years, when that grouping will recur,

(*c*) by taking any group of n consecutive birth-years and combining the experience in n-year groups. This will give a recurring pattern consisting of n^2 years of experience covering a range of $2n-1$ years grouped around a central year which contributes n years to the experience, and each year successively remote from this central year in each direction will contribute $n-1$, $n-2$ . . . . . $[n-(n-1)]$, *i.e.*1. This formula is general and Method (*a*) is in fact a special case of it, in which n is 1.

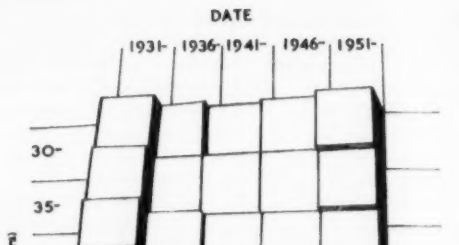

FIG. 3.—The age-specific death-rate grid considered as a surface.

From a practical point of view, Method (*a*) is not generally possible because of lack of data, and Method (*b*), used by Frost (1939) and Picken (1940), can be useful but is wasteful of data: a point which becomes important when studying mortality from individual causes of death. Method (*c*) is the one that has been most widely used, though none of the authors quoted has stated explicitly how the group experience was made up, and is the one which will be used in this paper: n has been set at 5 (quinary-quinquennial grouping), since this is the smallest age group used by the Registrar-General with sufficient consistency. The identifying birth-date of each cohort will be that of the central year, which will contribute 5 years of life experience to the quinary-quinquennial block and will not appear in any contiguous cohort.

When the mortality rates are arranged in this way, the grid shown in Fig. 1 may be considered as a surface, the third dimension being the age-specific death rate (Fig. 3).

This surface may be traversed in any of the three ways already described for the grid, but only the diagonal path will describe the "shadow of death" that is threatening the survivors of any one cohort on their march from the cradle to the grave.

Errors and inaccuracies in the vital statistics will affect the mortality rates that are entered in each square. Since the same entries in the squares are used, although in a different sequence, whichever way the grid is traversed, none of the three ways of traverse is unaffected by such errors. The error that might specifically affect the cohort method is that which has been introduced by emigration and immigration. Although this effect cannot be measured or corrected, it is not believed to have been serious in England and Wales during the period covered by these studies. In some countries this difficulty might prove an insuperable barrier to the use of cohort analysis. If this were to be so, no other method of traversing the surface could be relied on to make a reasonable biological statement about changing mortality patterns in that country.

The Underlying Concepts

We are now in a position to return to the introductory paragraph and to elaborate the ideas contained therein. A considerable part of the literature quoted has described cohort analysis from the actuarial standpoint, making the assumptions that the mortality curve changes smoothly with advancing age and that, if the curve can be graduated, it will be possible to extrapolate at least to a limited extent. The need for graduation and extrapolation has its roots in the demands of insurance companies for guidance in fixing insurance and annuity rates, and is not concerned with strictly biological considerations. In these circumstances it is but natural that actuarial writings are often irrelevant to questions that interest biologists.

Experience of the series of death rates traversed by the vertical lines (date-contours) had suggested that an equation of the general form $\mu x = \alpha + \beta c^x$ (Makeham, 1860) might adequately describe the shape of the curve traced by the force of mortality (μx) at any age x. As often happens, a convenient

shorthand description of shape was assumed by some to be a law of nature, and attempts were made to graduate or smooth the cohort mortality experience in order to find the terms of this equation and to extrapolate the curve from a "law of mortality". It may well be that there is in fact a hard core of mortality which improved environment and treatment will leave untouched; indeed, unless immortality is invoked, there must be some such core. It may also well be that this residual mortality will obey some mathematical law. It is very doubtful whether the mortality rates that have already been recorded approach this hypothetical "hard core". If they do not, the cohort mortality curves as they stand will be a better record of the effects of social history than the graduated curves.

The other pre-occupation of some of the authors quoted was that, whilst the pattern of mortality, either general or disease-specific, did in fact follow a pre-ordained course, even if not that prescribed by the Makeham type of formula, the intensity of the attack on the community varied from time to time. This variation was assumed to be fixed by the intensity of attack during the early years of life. The definition of the word "early" in this context varied from author to author.

Despite Karl Pearson (*e.g.* 1912) and his school, there is no reason to doubt that a record of early good health reflected by low death rates at early ages may in general predispose to continued good health reflected by lowered death rates at higher ages. At the same time it must be remembered that environmental and therapeutic changes occur continually during the life-span of each cohort. It is extremely likely that this continuous alteration will result in mortality changes other than those that can be attributed to influences at work in the early (by any definition) years of life.

If, then, cohort mortality can be fairly considered as the product of at least three factors:

(1) that which is biological and inescapable,
(2) that which is due to early nurture,
(3) that which is due to constantly changing environment and therapy (*i.e.* later nurture),

it follows that much of the previous use of cohort analysis has neglected to exploit its full possibilities, and has been based on an incomplete statement of the nature-nurture complex.

A profitable, and largely unexploited, use of the method would be to say: "The cohort analysis of a series of death rates shows certain changes over the years. The social history of the community concerned shows certain changes over the years. The changes in social history have led to the setting up of certain hypotheses linking them with changes in death rates. Are the changes in death rates revealed by the cohort analysis compatible with these hypotheses or not?". This would not be a short-term study and it would need a synthesis of the study of social conditions, the history of medicine and surgery, and of carefully compiled Tables of age-specific disease-specific death rates in a form suitable for cohort analysis. Haenszel and Shimkin (1956) have already conducted a limited investigation on these lines.

Some knowledge of the time-lag between putative cause and effect will also be required. If the cohort analysis of mortality rates is regarded as depicting ripples on the pond of human experience, and if these ripples are to be attributed to a particular brick being thrown into the pond, we must make quite certain they were not in fact there before the brick reached the surface of the water.

A necessary corollary to accepting the cohort analysis of a series of death rates as being the best biological interpretation of those given by the three methods of traverse is that we must abjure the vertical series (date-contour) as a basis for the formation of biological theories, unless the rates at each age over the whole series (age-contours) are constant, in which case the date-contours and the cohort series become identical. If enough data have been collected to investigate whether or not the rates at each age are constant, it is probable that enough data have been collected to make the cohort analysis. If they have not been collected, there is no logical justification for using the date-contour *faute de mieux*.

Examples of Results

General Mortality Rates in England and Wales

Figs 4A and 4B (overleaf) show the age-specific death rate per 1,000 living per year for males and for females.

Six cohorts, formed as previously described for quinary-quinquennial grouping, are used. The central years are spaced 20 years apart. The rates are calculated from populations and deaths that include non-civilians. The data are extracted from the successive volumes of what started as the "Annual Reports for the Years 1911–20" of the Registrar-General (1913–22) and continued as the "Statistical Reviews of England and Wales for 1921–54" (Registrar-General 1923–55). Data relating to the years before 1911 were abstracted from the "Annual Report" for 1911.

In general, each successive cohort shows a lower death-rate than its predecessors at all ages for which the rates have been calculated. In detail, this is not entirely true, because cohort 1891 shows a kink in both male and female mortality at age 25–29. This kink, which is of sufficient size to cross the line of cohort 1871 at that age, is due to the influenza epidemic of 1918–19.

The lowering of the mortality rates at later ages is clearly not entirely dependent on the lowering of mortality rates at ages under 5, for there is very little improvement at these ages between the cohorts

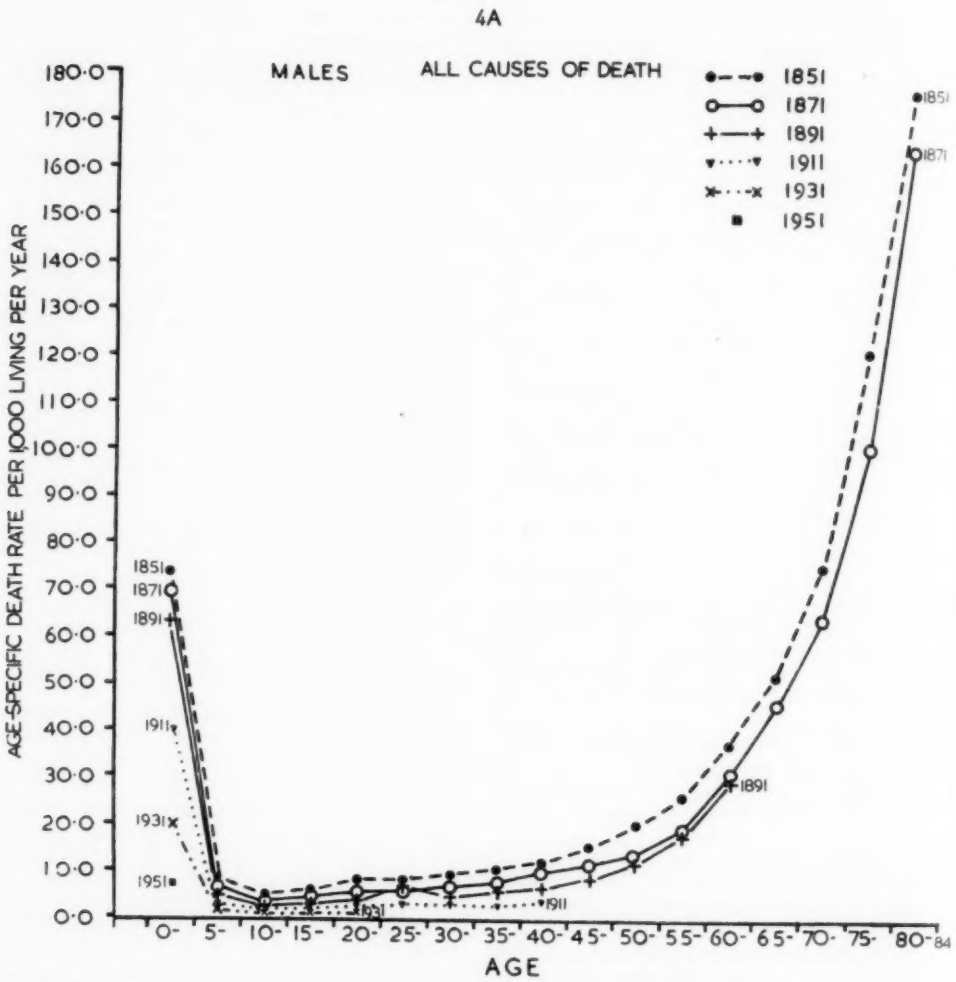

FIG. 4A.—Age-specific death rate in England and Wales (All Causes) per 1,000 living per year for the cohorts 1851, 1871, 1891, 1911, 1931 and 1951. Males.

1851, 1871, and 1891, whilst there is a marked improvement at, say, age 45 to 49. Infantile mortality (*i.e.* that under 1 year, not shown in the Figures) did not begin to drop until after 1901, but improvements at higher ages were established for cohorts born before then. Thus, whilst it is possible to deny the argument of Pearson (1912) that a lowering of the infantile mortality rate must be inevitably followed by raised mortality rates in the juvenile range, it is not possible to invert this argument, and to say that improvements in mortality rates at any age must necessarily be preceded by improvements in the mortality rates at an earlier age in respect of any two cohorts being compared.

In Figs 5A and 5B (overleaf), cohorts spaced less widely have been used, though not all are drawn over the whole extent of their courses lest graphical confusion result. The effect of the 1918–19 influenza epidemic can now be seen as a kink at age 15–19 in the 1901 cohort, at 25–29 in the 1891 cohort, and at 35–39 in the 1881 cohort. It is still detachable, though masked by the rapidly rising force of mortality, at age 45–49 in the 1871 cohort. The persistence of this kink in both the male and female

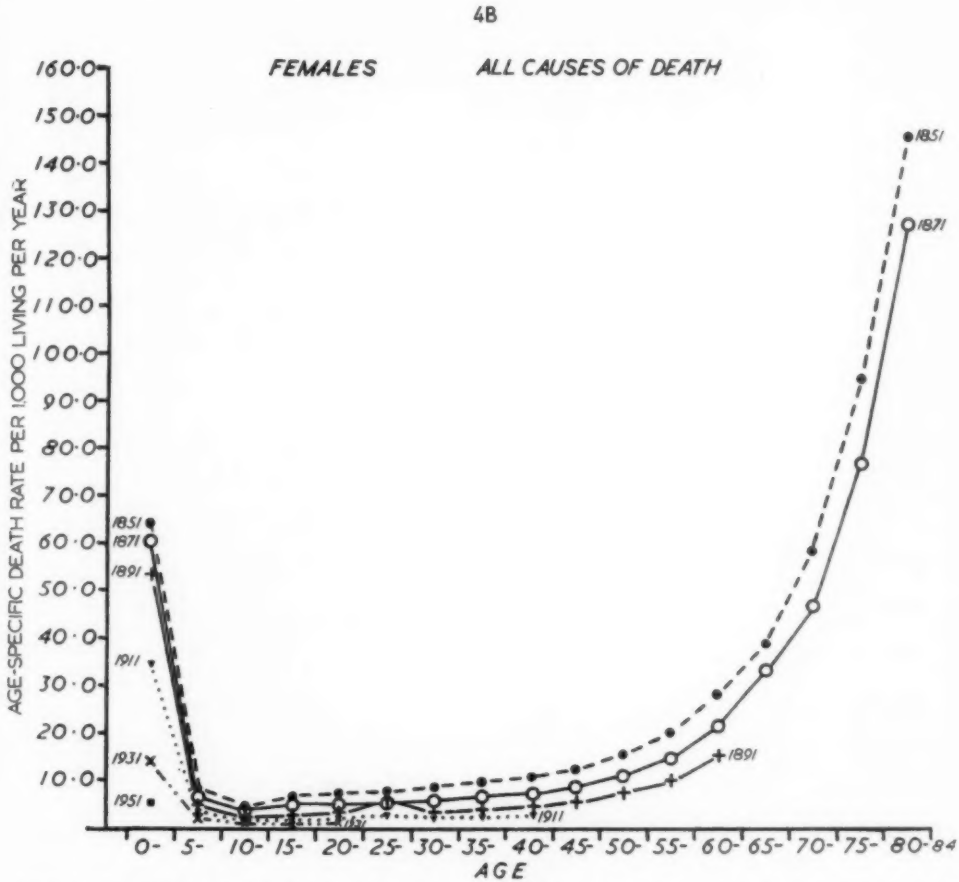

FIG. 4B.—Age-specific death rate in England and Wales (All Causes) per 1,000 living per year for the cohorts 1851, 1871, 1891, 1931, 1931 and 1951. Females.

cohorts differentiates it from an increased mortality affecting men who served in the 1914–18 war. The use of a quinquennial group underestimates the true severity of the epidemic. This detailed analysis also shows that several of the male cohort lines overlap at higher ages, or show but little improvement; this tendency is hardly to be seen amongst the female cohorts.

Thus, whilst a change in mortality has taken place during the last century, this change has not been entirely predetermined by the mortality of the earliest years of life, nor has it always been constant

5A

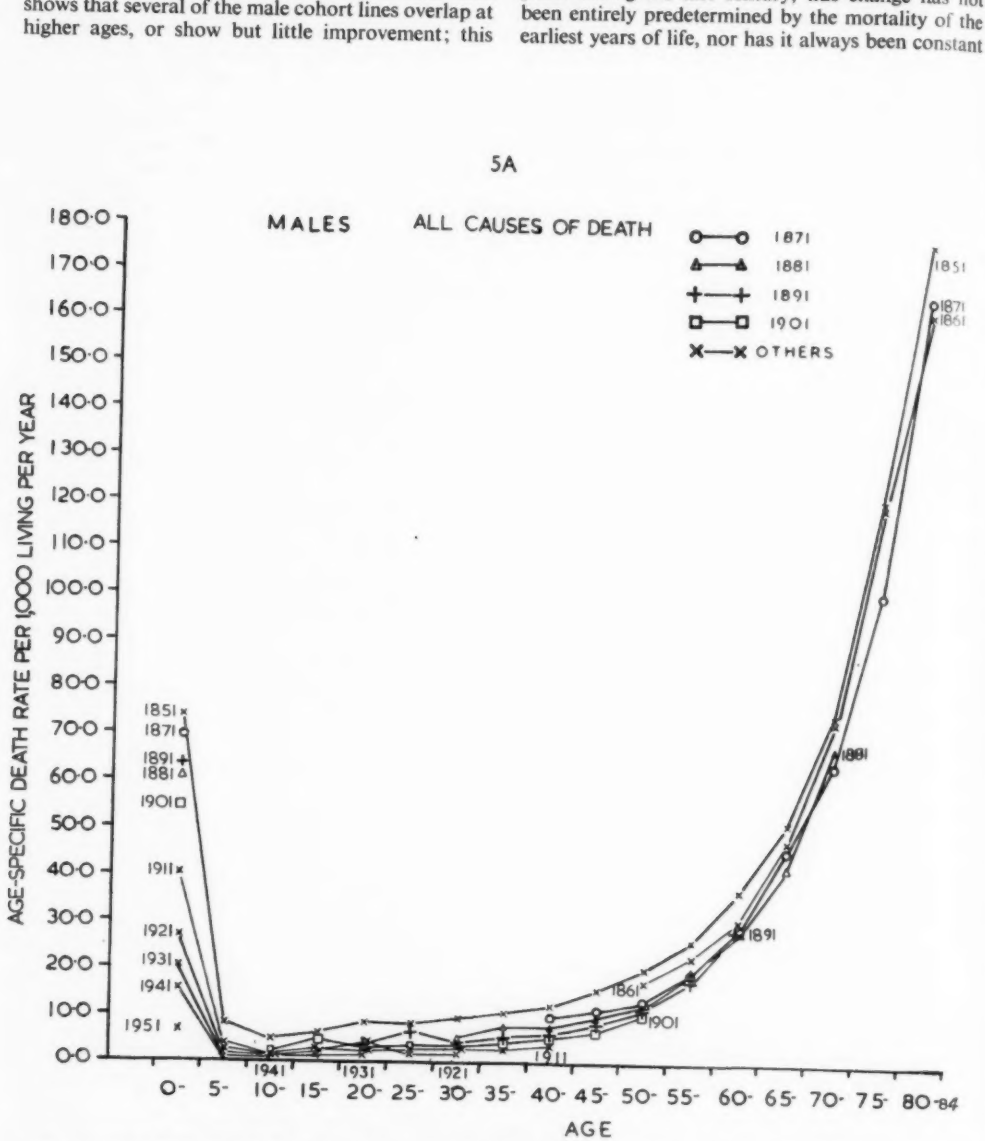

FIG. 5A.—Age-specific death rate in England and Wales (All Causes) per 1,000 living per year for the cohorts 1871, 1881, 1891, 1901; with some additional detail from the cohorts 1851, 1911, 1921, 1931, 1941, and 1951. Males.

in direction. Although man is unique amongst animals in being able to generate intentionally a constantly changing environment, he is not prescient, and there is no reason to believe that his changes will always be beneficial. There is therefore no compelling reason arising from these studies to believe that the occasional deterioration in the male death rates at higher ages is a reflection of the late action of deleterious genes or is otherwise determined by failure of selection at earlier ages, since an adverse adult environment of relatively recent origin could also produce this change. There is also no compelling reason to reject the former hypothesis; elucidation must be sought elsewhere.

In most of what has gone before, the discussion has been confined to general mortality. When individual causes of death are considered, there is even less reason to believe that for many diseases the "hard core" of the mortality pattern has been reached. How far early environment will be effective in fixing the mortality pattern, and how far later environment and therapy will affect this pattern will vary considerably from disease to disease. The picture revealed by cohort analysis for different diseases must be assessed in the light of what is known about each one.

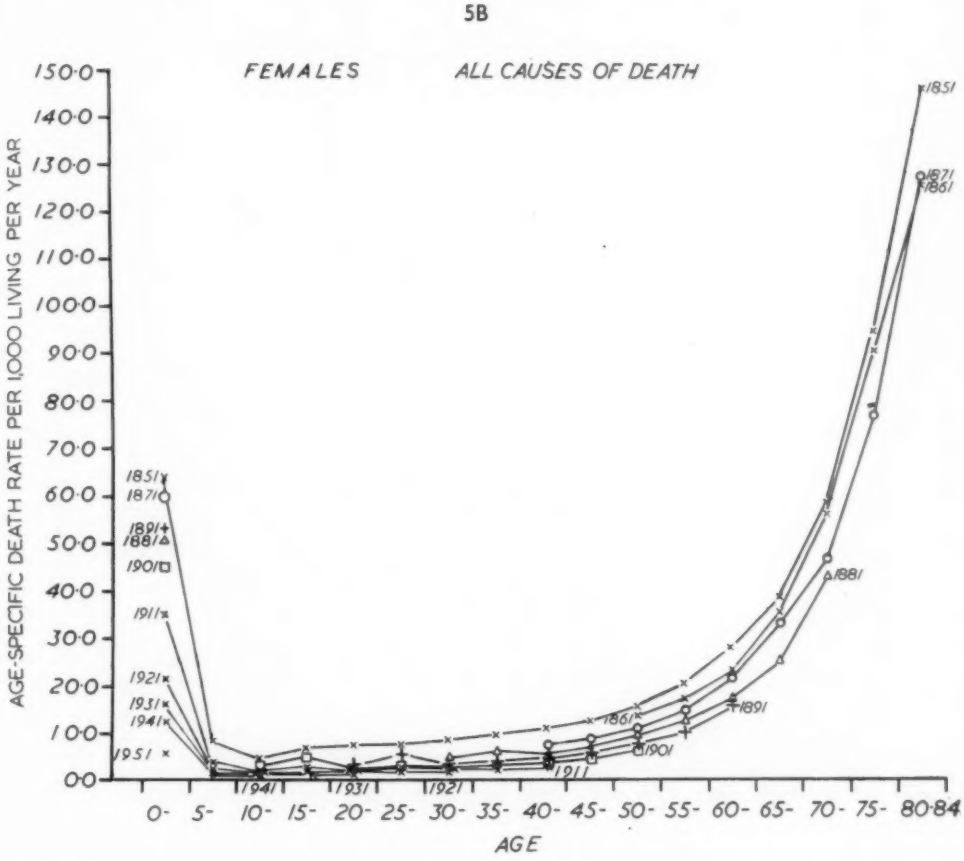

FIG. 5B.—Age-specific death rate in England and Wales (All Causes) per 1,000 living per year for the cohorts 1871, 1881, 1891, 1901; with some additional detail from the cohorts 1851, 1911, 1921, 1931, 1941, and 1951. Females.

EFFECT OF A CHANGING RATE ON THE MORTALITY PATTERN AS SHOWN BY COHORT ANALYSIS AND DATE-CONTOUR ANALYSIS

The interpretation of mortality patterns revealed by date-contour anlysis can be made more simple, and certain pitfalls can be avoided, if the effects of a changing rate are realized. The examples shown here are taken from the cancer field, not only because one of them has already been discussed by Korteweg (1951, 1953), but also because the relatively simple form of the mortality pattern for cancer, and the relatively slow fluctuations that are exhibited, make these illustrations almost as simple as one could obtain by using hypothetical data.

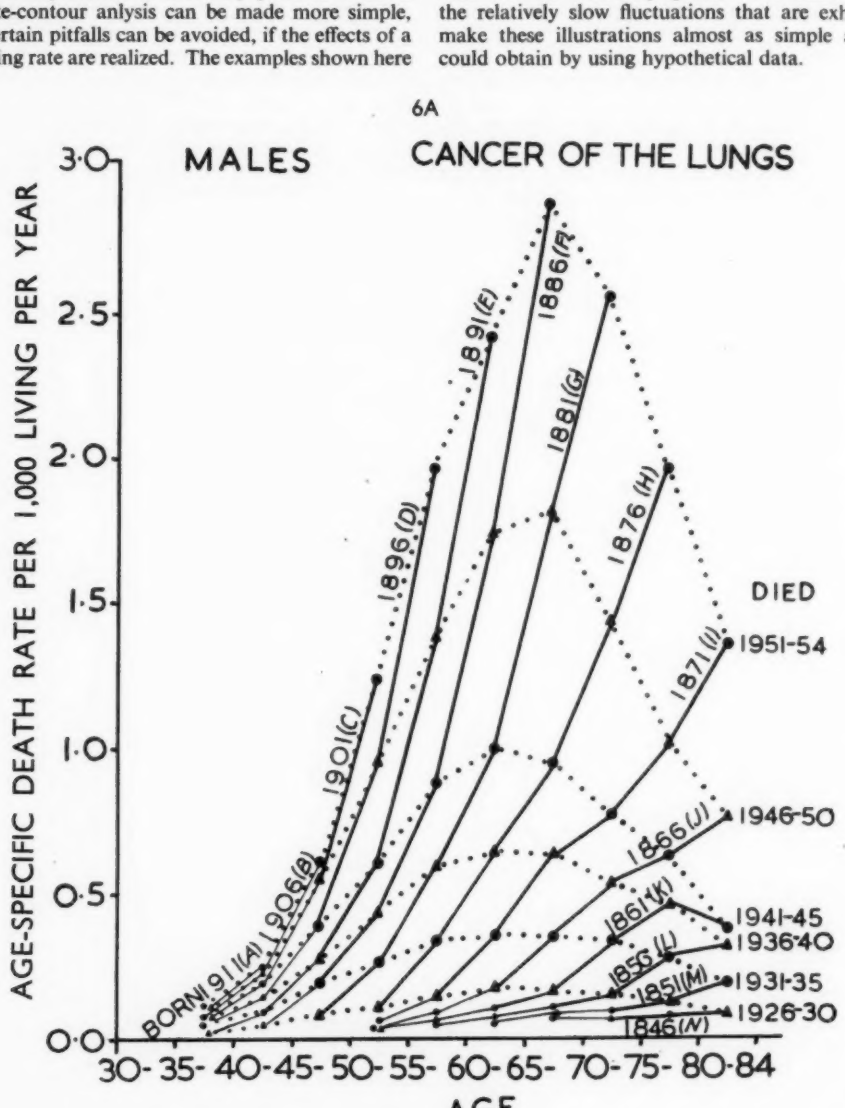

FIG. 6A.—Cohort and date-contour analysis of changing rate. Age-specific death rate in England and Wales (Cancer of the Lungs) per 1,000 living per year, in quinary-quinquennial groups from 1846 to 1911, arranged according to date of birth (cohort analysis) and date of death (date-contour analysis) for males. The rapid rise exceeds the critical level.

(1) *Effect of a Rising Rate.*—Figs 6A and 6B show all the squares of the quinary-quinquennial grid plotted and joined along the cohort line and along the date-contour line for the recorded mortality for cancer of the lung in England and Wales from 1911 to 1954.

In males the date-contour line shows the highest death rates to lie between the ages of 60 and 70, but the cohort line shows a continuous rise. This effect will always be produced when the rise from date-contour to date-contour exceeds a critical ratio to the rise from age group to age group in each cohort. This is the effect described by Korteweg (1951, 1953).

In females the date-contour line flattens out and

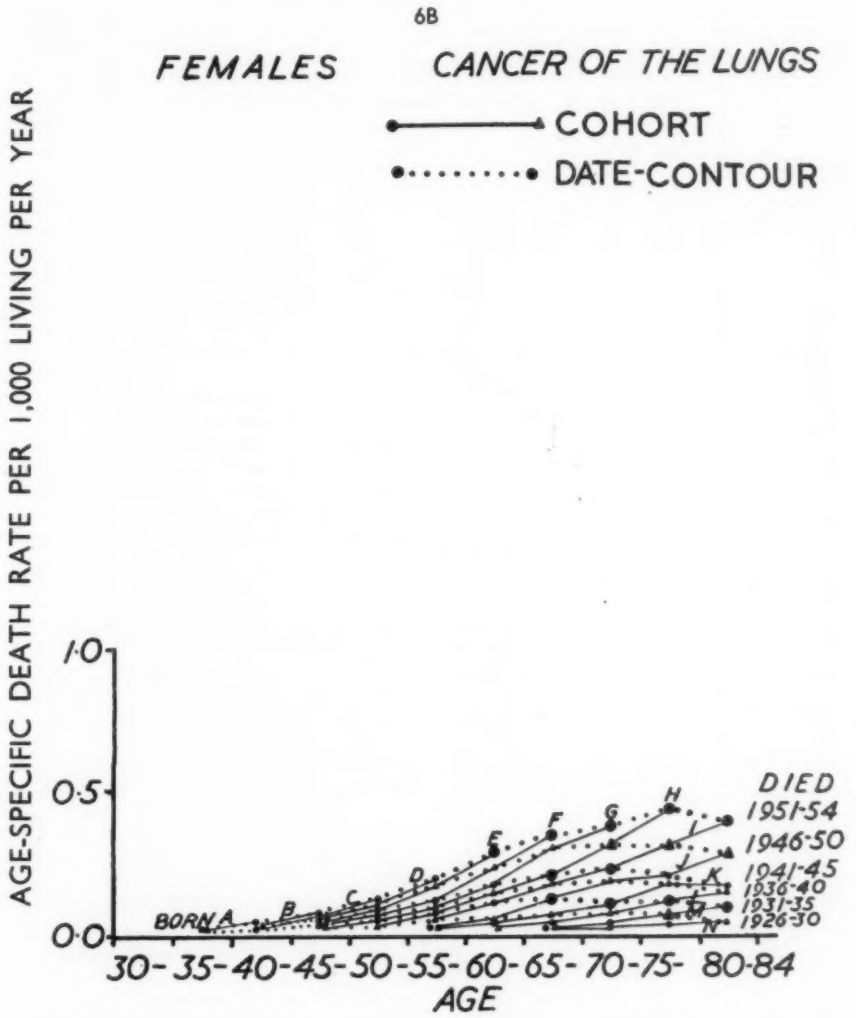

FIG. 6a.—Cohort and date-contour analysis of changing rate. Age-specific death rate in England and Wales (Cancer of the Lungs) per 1,000 living per year, in quinary-quinquennial groups from 1846 to 1911, arranged according to date of birth (cohort analysis) and date of death (date-contour analysis) for females. The slower rise just attains the critical level.

shows an apparent lack of age specificity, but the cohort line reveals a ready rise. In this example the rise from contour to contour is only just attaining the critical level required to produce an apparent modality in the date-contour line.

(2) *Effect of a Falling Rate.*—Fig. 7, illustrating the mortality from cancer of the lip for males in England and Wales, shows the effect of a falling rate. The date-contour line shows a hollowing out, giving an inflection in the middle of the range which is not found in the cohort lines.

If we wish to consider the effect of a rising or

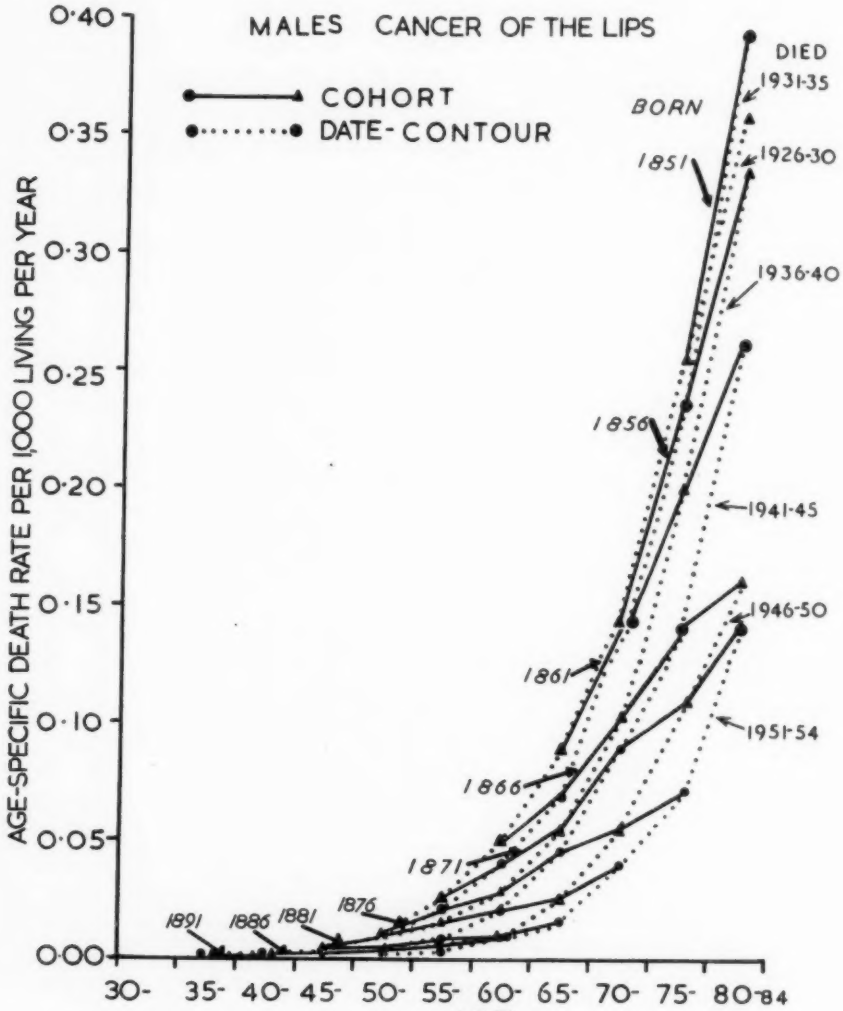

FIG. 7.—Cohort and date-contour analysis of changing rate. Age-specific death rate in England and Wales (Cancer of the Lips) per 1,000 living per year, in quinary-quinquennial groups from 1851 to 1891, arranged according to date of birth (cohort analysis) and date of death (date-contour analysis) for males. The rapid fall produces a marked inflexion in the date-contour line.

falling rate on more complex curves, such as the modal type of curve shown by tuberculosis mortality (Springett, 1950), the same principles of curve analysis can be applied to each segment.

These two illustrations show how important it is not to base biological theories on the shape of the mortality curve derived from the date-contour unless it is known that the rates have been substantially unchanged during the time covered by the age range under consideration.

An example of such a theory, recently advanced, is that of Nordling (1953), who uses the date-contour of the death rates from cancer of all sites to suggest that, since this yields a log-logarithmic plot reasonably described by the exponential of the sixth power, cancer might be the result of a seventh mutation. Stocks (1953 a, b) recognizes that such a theory should be based on cohort analysis, but does not explore the cancer field sufficiently widely to demonstrate that, if such a theory could be based on death rates, it must apply to only a few sites of cancer, or that the theory must be so qualified that the postulated number of mutations would vary not only from site to site, but also for the same site at different times.

It is not the author's intention to deny that any hypothesis invoking a sequence of changes or mutations as a necessary precursor to malignant change could be valid, but only to point out that premises based in this way on the date-contour analysis of mortality rates offer at the best a very insecure foundation for advancing such a hypothesis.

Summary

A history of cohort analysis has been given, and it has been pointed out that the bulk of the literature on the subject has dealt with concepts that assume *either* that graduation and possibly extrapolation are desirable, preferably in conformity with some formula expressing what is implicitly accepted as a "law of mortality", *or* that, whether or not a fixed pattern of mortality exists, the intensity of mortality risk is largely determined in early life.

The view is advanced that either concept alone is, or both concepts together are, inadequate and may lead to an improper assessment of the nature-nurture complex, since environment and therapeutic measures are constantly changing. The plea is made for the technique of cohort analysis to be used as a narrative or historical technique, and for a synthesis of knowledge derived from social history, medical history, and cohort analysis to be made to interpret the narrative.

The effects of a changing rate on the mortality pattern as depicted by date-contour analysis and cohort analysis are outlined and the danger of basing biological theories on the mortality pattern revealed by date-contour analysis alone is indicated. This point is illustrated by reference to a recent theory that has been based on such an analysis.

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COHORT ANALYSIS OF CANCER MORTALITY IN ENGLAND AND WALES, 1911–1954 BY SITE AND SEX

BY

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Introduction

Considerable confusion seems to exist amongst both medical and lay people about current trends of cancer mortality. This is hardly diminished by Raven (1955) when he confuses *number* and *rate* so much that he follows his statement "It is frequently asked whether or not the death-rate from cancer is increasing" by quoting the *number* of deaths from cancer during the years 1943 to 1953 and commenting "The figures show the steady increase in the *death-rate* from cancer in this ten-year period." [*Italics* inserted]. It therefore seems an appropriate time to use the narrative technique of cohort analysis to show what has happened to the mortality rates, male and female, in England and Wales since 1911.

A few authors have already published cohort studies on some aspect of mortality or morbidity from cancer of the lung (Korteweg, 1951, 1953; Clemmesen, Nielsen, and Jensen, 1953; Levin, 1953; Stocks, 1953a; Cutler and Loveland, 1954; Haenszael and Shimkin, 1956). Cohort analysis is mentioned by Dorn (1953) and a diagram of cohort analysis of male mortality from cancer of the lung in England and Wales is given without comment or description by Kennaway and Waller (1953). Stocks (1953a, b) also shows cohort analysis figures for cancer of the female breast, the uterus, and the stomach, but obscures for many readers the narrative properties of the method by using a semi-logarithmic plot. Hewitt (1955) analyses leukemia mortality by the cohort method.

No author seems to have attempted a systematic survey of cancer by site for any country, and none of the authors cited has given sufficient description of the principles involved in the method either to enable the non-statistical reader to follow it readily or to convince the methodologist of the validity of its logical credentials. This has been attempted in a previous paper (Case, 1956).

The present studies attempt in general to outline the changes that have taken place in cancer mortality in England and Wales for 21 different sites of cancer in the last 45 years, and in particular to answer three questions, posed in a biological form, because they still seem to be of great interest to many workers in the field of cancer research as well as to clinicians. These questions are:

(1) Is the lethal force with which cancer affects the community increasing and if so what forms of cancer contribute to this increase?

(2) Is there, generally speaking, a "cancer-age" or does the force of mortality due to cancer at different sites increase throughout the life span?

(3) Is there a consistent sex-difference in the force of mortality due to cancer at the different sites that are common to both sexes, and if so how great is this difference?

No claim is made that these questions are not answered elsewhere, or that they could not be answered by some other method of analysis, although the author believes that no other method has answered them in a way so readily understood by readers with a modicum of biological insight but no special statistical knowledge.

The studies will immediately suggest many other questions to which no answer has been hazarded. This is because the results of cohort analysis are only one component of the synthesis of knowledge required for answering such questions. This topic has been discussed more fully elsewhere (Case, 1956).

Materials and Methods

The previous paper has explained how the essential feature of cohort analysis is that it follows the mortality rate of a population defined by its birth years throughout the lifetime of the surviving portion of that population, thus offering some chance of distinguishing between the

effects of nature and of nurture on the death rates, and also making it possible to correlate the changes in the death rates with the social history of the period.

In order to provide sensitive and comprehensive methods for detection of environmental cancer risks, Case and Pearson (1955) had undertaken firstly a concordance of the Cancer Mortality Tables given in the "Annual Reports of the Registrar-General" (1911–20) and the "Registrar-General's Statistical Reviews of England and Wales" (1921–54), and secondly the systematic calculation of age-specific death rates for cancer at 21 different sites for each sex in quinary-quinquennial groups from 1911 onwards. Similar Mortality Tables were computed for All Causes of Death; Leukaemia; Hodgkin's Disease; Benign Tumours and Tumours of Undetermined Nature; and Cancer at All Sites. A further Table for cancer at sites other than the 21 specified ones was obtained by difference.

Many of the source Tables used departed from quinary grouping at ages under 35, and although Case and Pearson (1955) were able to interpolate sufficiently accurately for environmental analysis below this age, the cohort analyses used in these studies are confined to the age range 30 to 84 except in the case of Leukaemia and Hodgkin's Disease, where the source Tables are consistently divided into quinary groups. Although the concordance enabled the classification to 21 sites defined in 1911 to be followed throughout the years, certain changes in the way of classifying the cause of death to which a death certificate was assigned were introduced in 1939. The information published by the Registrar-General (1944) in the "Statistical Review" for 1939 has allowed the calculation of a correction factor for many sites, indicated in Appendix Table I, but there remains a possibility of a discontinuity in rates around 1940. This will be most marked in the older age groups.

The rates have always been calculated from figures that include non-civilians, for there is a marked difference in the rates for civilians and non-civilians, doubtless due to selection of fit subjects and the discharge of cancer patients from the services.

A variation in one disease-specific age-specific death rate does not compel any other disease-specific age-specific rate to alter; indeed, such a concomitant change will take place only if there is a synergism or antagonism between the lethality of the two diseases. The mortality from All Causes is, as the name implies, the sum of all the disease-specific death rates for the relevant age and date.

The rates in the Tables form a domain for exploration and investigation of the mortality trends. The extent of this domain, and the main paths of exploration, are shown in Fig. 1.

Four main cohorts, formed by quinary-quinquennial grouping as described by Case (1956), are used consistently throughout the analysis. These cohorts were born around the central years 1871, 1881, 1891, and 1901; they are labelled with these dates and also with a distinctive symbol which is used to plot each value (Fig. 1).

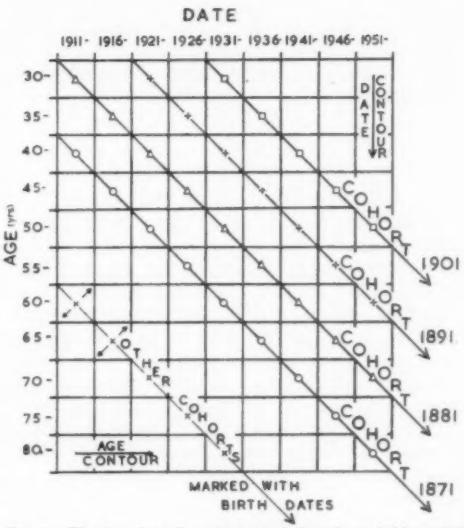


FIG. 1.—The domain to be explored and the cohorts (with symbols) used in the exploration.

These particular cohorts are chosen for the following reasons:

(1) The 1871 cohort attained the 80–84 age group in 1951–55, and thus became statistically extinct, passing into the indeterminate age group 85 and over for which no meaningful death rate can be computed.

(2) A period of 10 years between the cohorts is a convenient interval since it does not obscure the picture with too much detail, as the consistent use of a 5-year interval would do.

(3) The age group 50–54 is about the lowest at which analysis of the trend is profitable, and this is the age group attained by the 1901 cohort in the last quinquennium of the domain.

Sometimes it is desirable to use another cohort to help to elucidate some feature of interest in a particular picture. When this is done the extra cohort, which uses a symbol common to all such extra cohorts, is marked with the date of the central year of birth.

All death rates are plotted as rates per 1,000 living per year, and no logarithmic transformation has been made lest this obscure the narrative property of the method for readers who are not familiar with such a transformation.

It is of course a commonplace that death certificates are not an accurate measure of either mortality or morbidity from particular causes. The errors involved have been discussed at length by Kennaway and Kennaway (1936) and by Willis (1953). They will apply to any method of analysing age-specific death rates, and are not a special complication of cohort analysis.

The use of morbidity data for England and Wales is impossible because such records are not available in

sufficient detail, and even if the Cancer Registration Scheme described by the Registrar-General (1955) could now be considered perfect, 45 years would have to elapse before a morbidity analysis comparable with this mortality analysis could be made.

Results

The results of these studies are set out in detail in Appendix Table I. They can fortunately be presented much more clearly in graphical than in textual form. The reader is therefore asked to study each figure in conjunction with the relevant text.

Fig. 2 (opposite) shows how the mortality rate from cancer at ages over 45 fits into the general mortality pattern of the four main cohorts. It also shows the proportion if the cancer mortality attributable to certain large groups of sites: Lung; Gut from cardia to anus, subdivided into Stomach, Intestines, and Rectum; Sexual Sites, subdivided into Uterus, Breast, and Others in the female, and into Prostate and Others in the male; and the aggregate of sites not included in these categories.

The picture for the 1871 cohort can be drawn up to the age of 85, but ... cohort 1881 had only reached the age of 70–74 in the final quinquennium of the domain, and so the picture must finish at that age. For each succeeding cohort the picture will finish progressively 10 years earlier. To make comparison easy, a strip from the preceding cohort or cohorts showing their pattern of mortality at the relevant age has been included in each picture.

In all the cohorts the male general mortality always exceeds the female general mortality, and the male cancer mortality exceeds the female cancer mortality at all ages above 55. The general mortality is decreasing at all ages shown for females, and at all ages under 60 for males. The male 1891 cohort has an unfavourable mortality at age 60–64 compared with the 1881 cohort, though still favourable when compared with the 1871 cohort.

The cancer mortality for males increases in each successive cohort, whilst that for females decreases. The most dramatic feature of the cancer mortality broken down into the large subdivisions is the increase in mortality from cancer of the Lungs in males. In the 1871 cohort cancer of the Lungs was never a major item in the balance sheet. In the 1901 cohort at the age of 50–54, cancer of the Lungs accounts for about half of the total cancer mortality rate and for about one-eighth of the total mortality rate. In the female the rate for cancer of the Lungs though increasing, has not yet assumed major importance. The mortality rate from all forms of cancer other than cancer of the Lungs shows a progressive decrease in each succeeding cohort in both sexes, but the form of presentation using grouped sites does not throw much light on how this comes about.

The increase in the male mortality from cancer of the Lungs from the 1881 cohort to the 1891 cohort at the age of 60–64 is in itself sufficiently great to account for the increase in general mortality already discussed at that age. This is not to say that it is in fact solely responsible for this increase.

Part of the information gained from Fig. 2 is also presented in another form, which will be used consistently throughout the succeeding analyses. Fig. 3 (overleaf) shows the mortality rates for Cancer at All Sites for all the four cohorts drawn from the same origin on the age scale. Where such a plot results in lines running together, the cohort symbols are plotted on the horizontal level of the rate, though in some cases they are displaced laterally. The line joining the centre point of such symbols is broken into as many segments as there are lines run together. The advantage of this form of plotting is that it is simple to compare the mortality rate of a cohort at a particular age with that of the other cohorts when they were at that age.

Fig. 3 shows that the male cancer mortality rate for each successive cohort exceeds that of the preceding cohort, and that at age 50–54, the most advanced age where all four cohorts are depicted, the mortality rate of the 1901 cohort is 122 per cent. of that of the 1871 cohort at the same age. (Appendix Table II sets out this proportional change for each cohort at each site). The female cancer mortality rate has decreased in each successive cohort, and the mortality of the 1901 cohort at age 50–54 is only 82 per cent. of that of the 1871 cohort at that age.

Fig. 4 (overleaf) shows the mortality rates for Cancer at All Sites except the Lungs. In both males and females there is now a progressive diminution in the rate for each successive cohort, the male rate for the 1901 cohort at age 50–54 being 68 per cent. of that for the 1871 cohort at that age, and the female rate 78 per cent.

The justification for analysing the cancer death rates with and without the contribution due to mortality ascribed to Cancer of the Lungs is that contemporary thought is sharply divided about the reason for the recorded increase of the mortality from lung cancer.

One school maintains that the increase is almost entirely spurious, being the result of improved diagnosis. If this were true, since it would seem that the misdiagnosed cases would previously have been

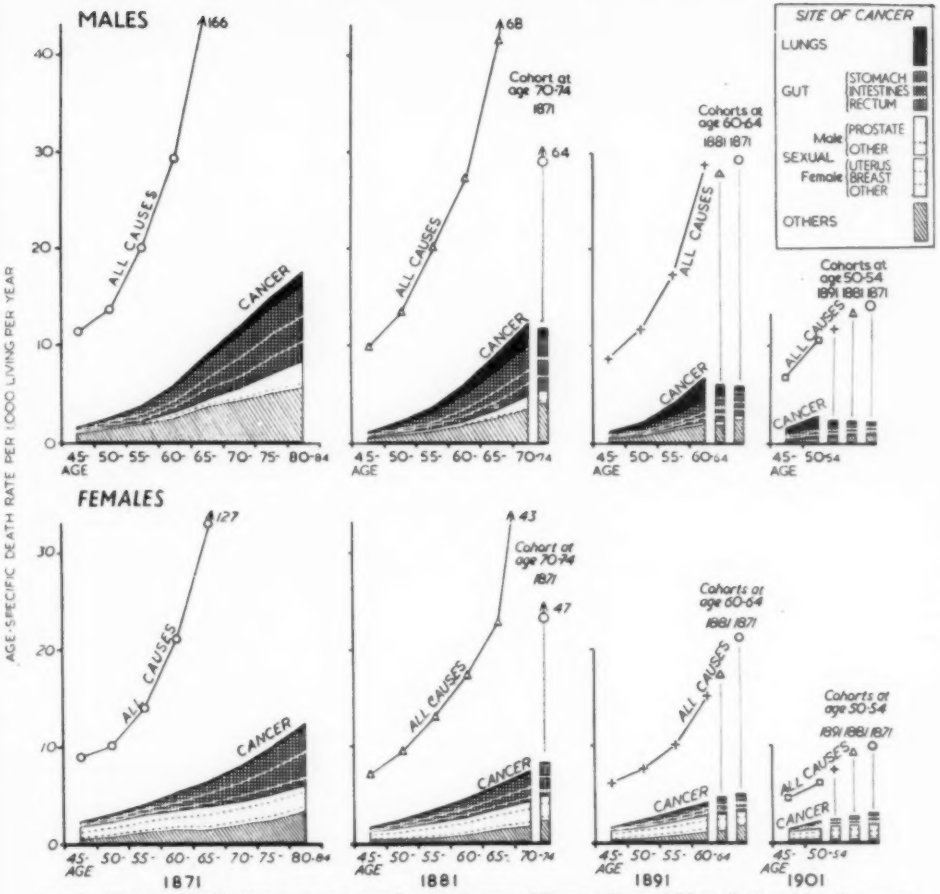


FIG. 2—The age-specific death rate picture for All Causes of Death (General Mortality) and for Cancer at All Sites. The proportionate contributions of the main sites are also shown.

allocated to some disease category other than cancer, a progressively larger amount must be added to the cancer mortality rate of each cohort as the birth-date grows more remote. The final picture would then show a progressively diminishing cancer mortality, as in the female.

The other school, whilst not denying that diagnostic changes have taken place, asserts that a considerable part of the increase is due to environmental influences under human control. If this view is correct, the remedy lies in removing these influences. The interest in cancer mortality then becomes transferred to the residual sites of cancer, where as yet we cannot make any confident assertion about the causation of any large fraction of the mortality experience.

Figs 5 to 28 show cancer mortality at 21 different sites for each sex, three sites being sexually unique (Ovaries and Fallopian Tubes, Uterus, and Vulva and Vagina in the female; Testes, Prostate, and Penis and Scrotum in the male). Some of the site designations may well be considered quaint by present-day standards (*e.g.* Kidneys and Adrenals), but it was necessary to use the 1911 site classification; for, although it was possible to combine the more detailed groups that later made their appearance to give the original groupings, there was no way of subdividing the earlier omnibus headings.

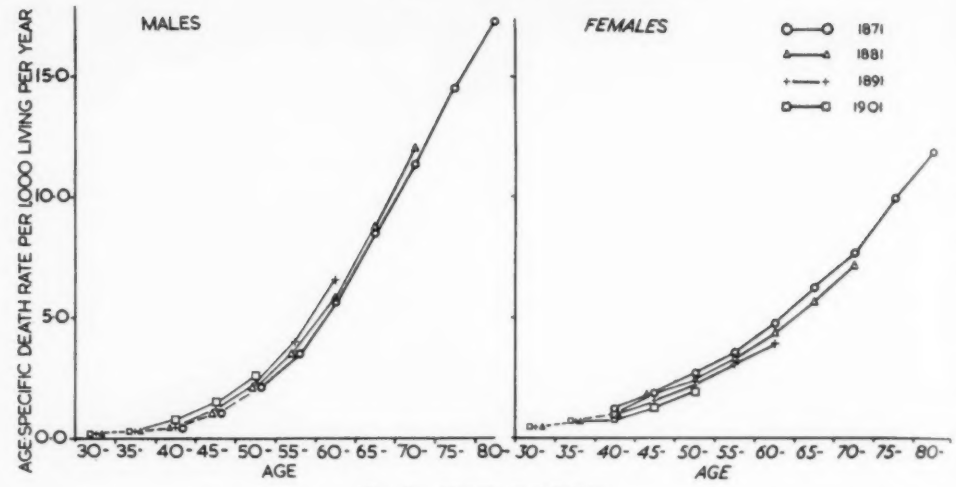


FIG. 3.—Cancer at All Sites.

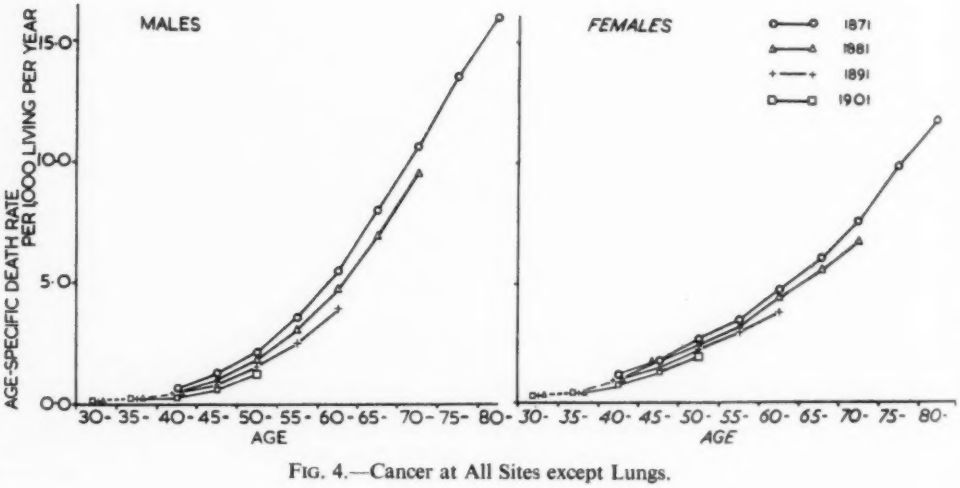


FIG. 4.—Cancer at All Sites except Lungs.

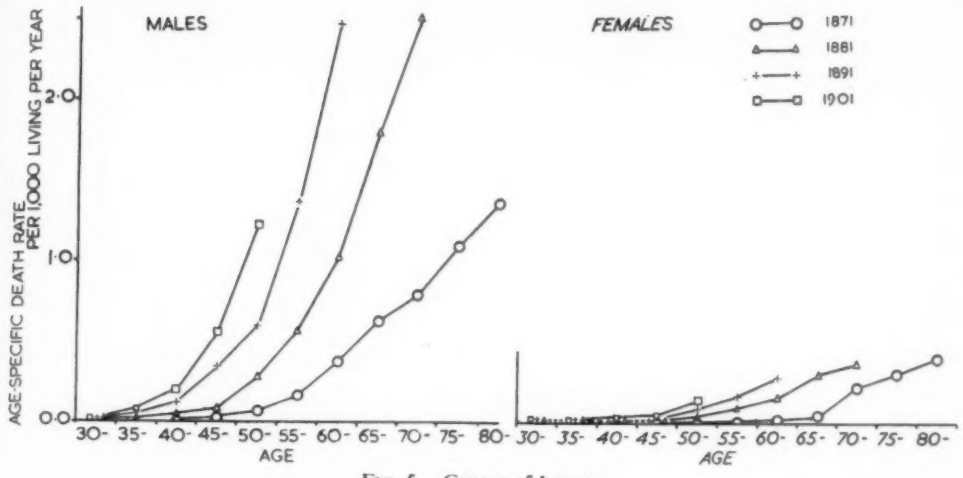


FIG. 5.—Cancer of Lungs.

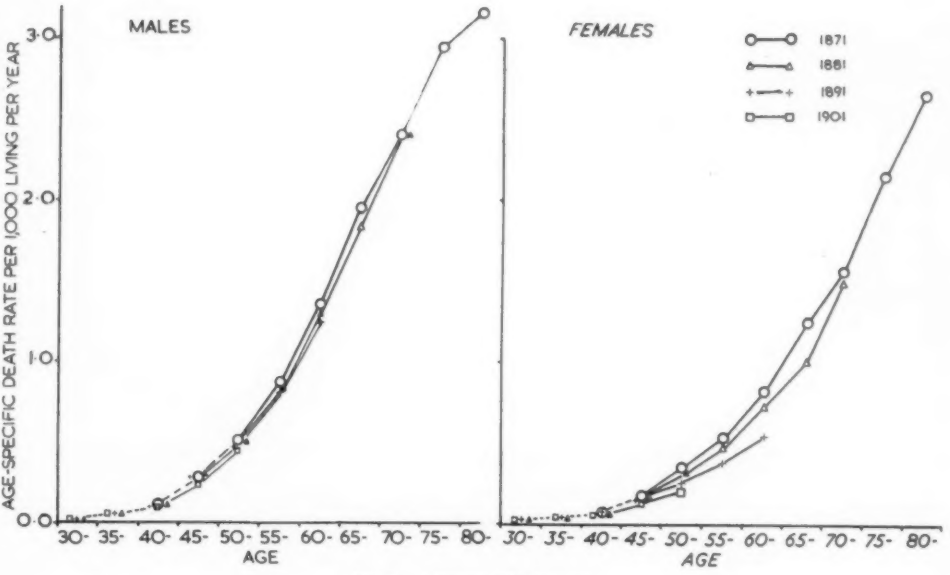


FIG. 6.—Cancer of Stomach.

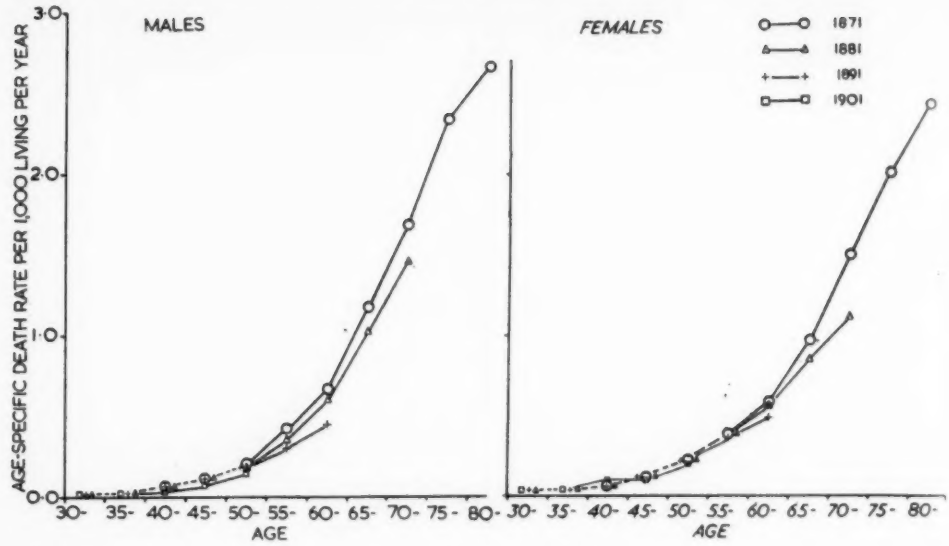


FIG. 7.—Cancer of Intestines.

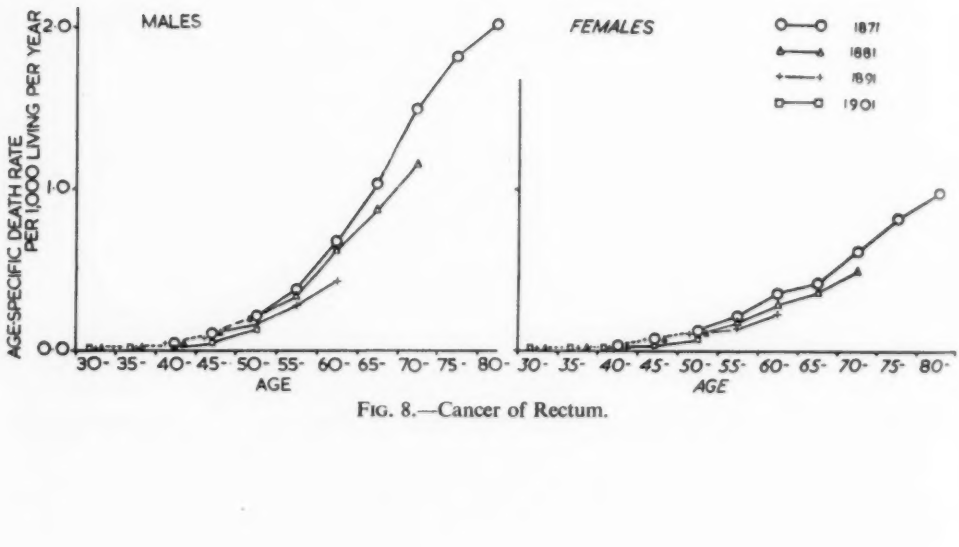


FIG. 8.—Cancer of Rectum.

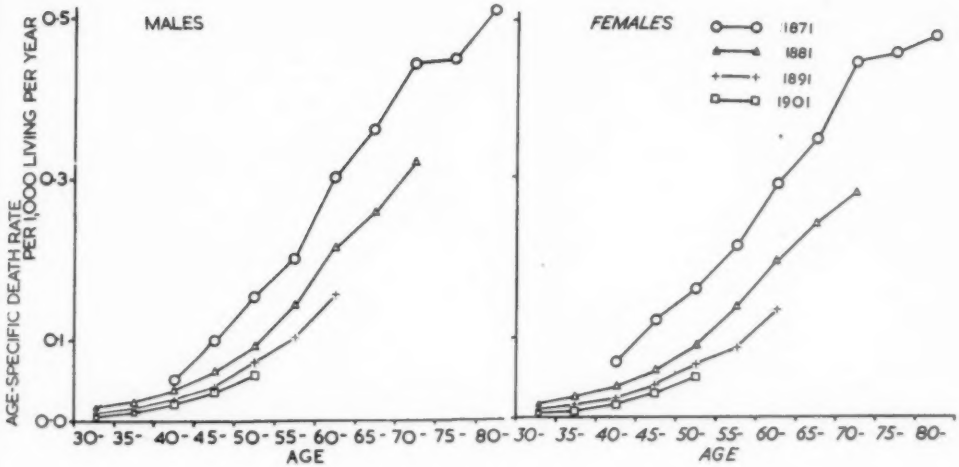


FIG. 9.—Cancer of Liver and Gall Bladder.

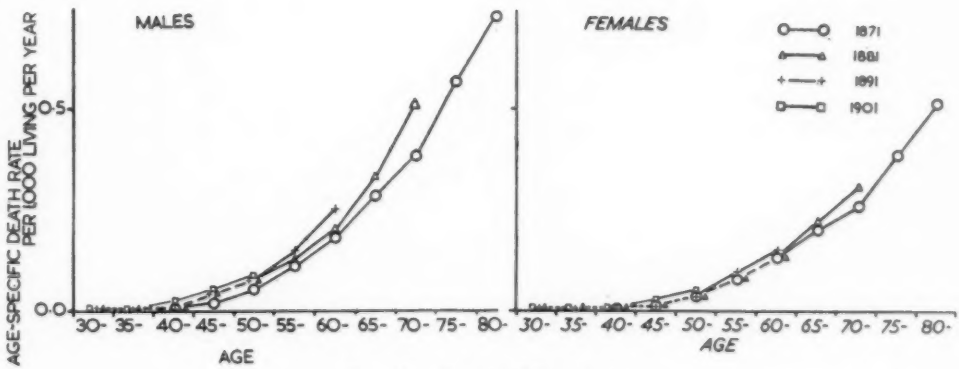


FIG. 10.—Cancer of Pancreas.

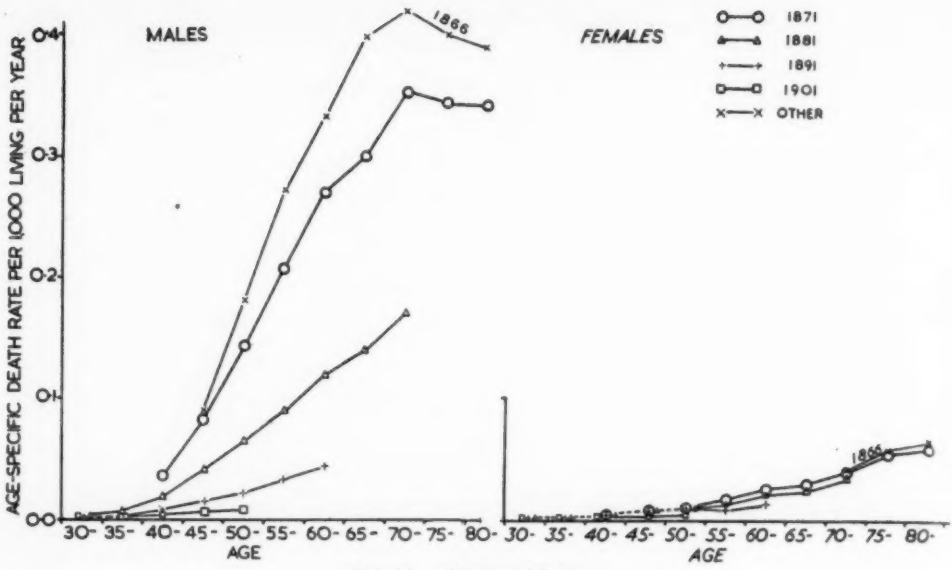


FIG. 11.—Cancer of Tongue.

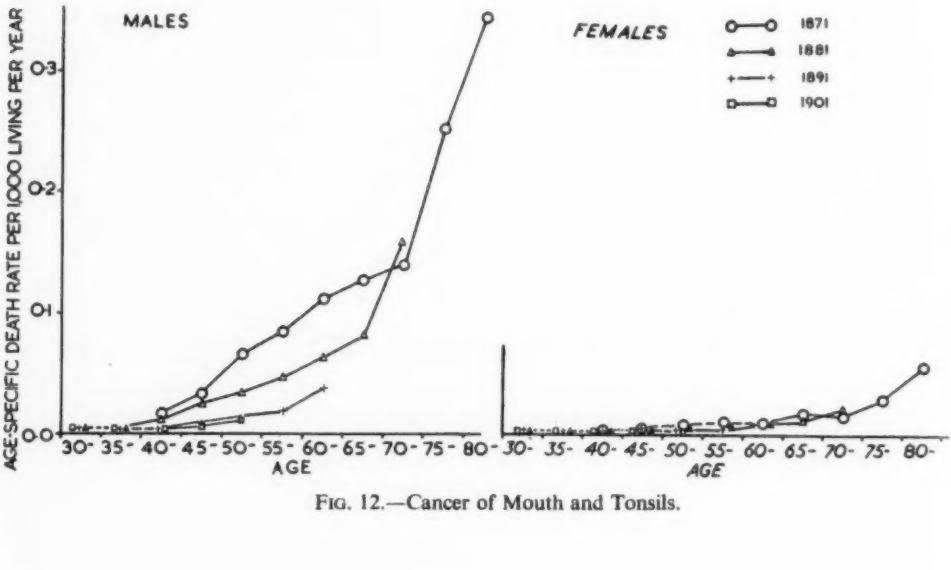


FIG. 12.—Cancer of Mouth and Tonsils.

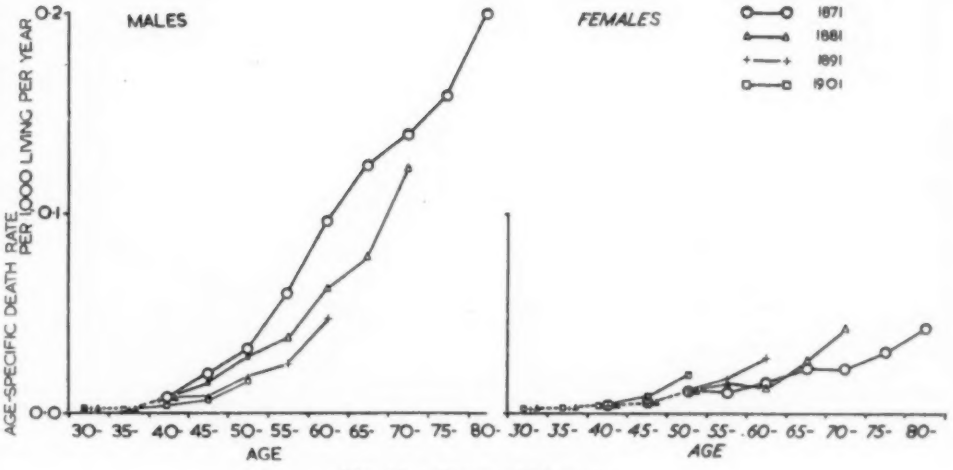


FIG. 13.—Cancer of Pharynx.

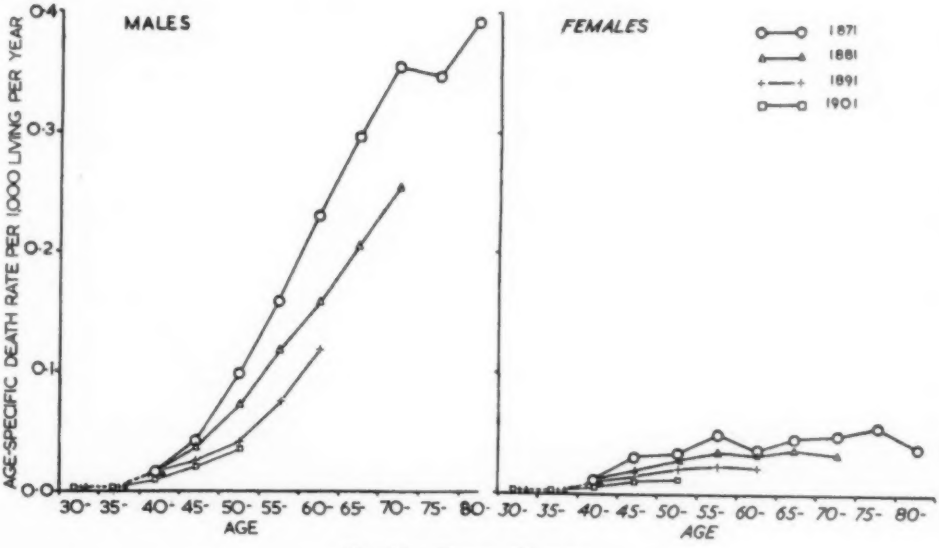


FIG. 14.—Cancer of Larynx.

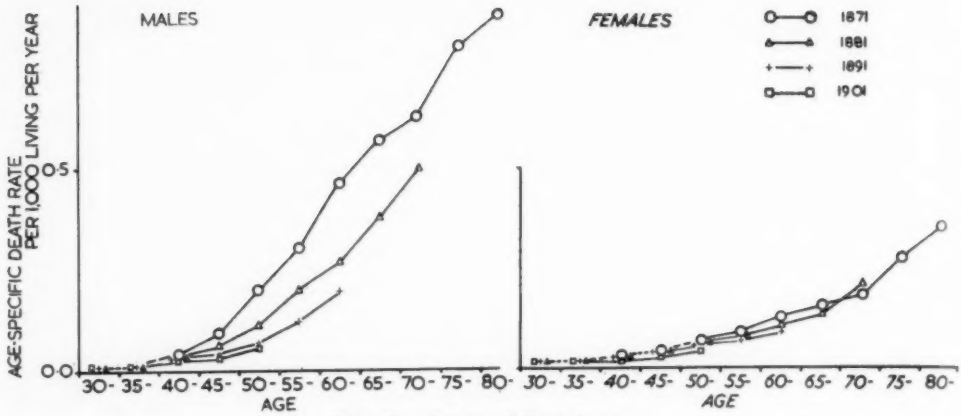


FIG. 15.—Cancer of Oesophagus.

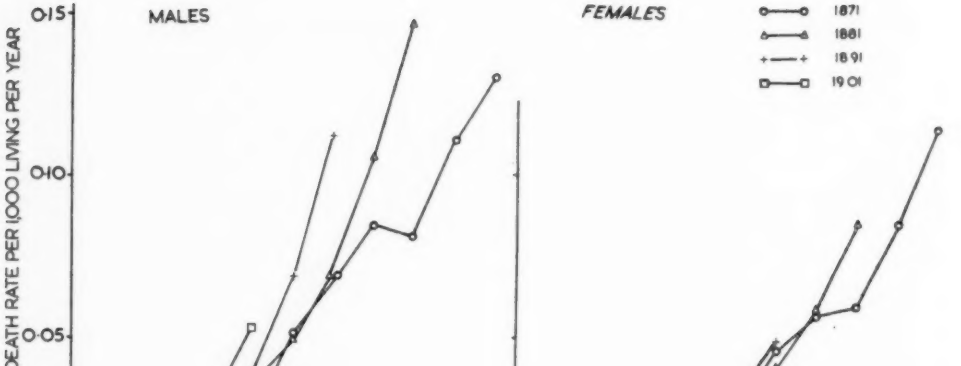


FIG. 16.—Cancer of Kidneys and Adrenals.

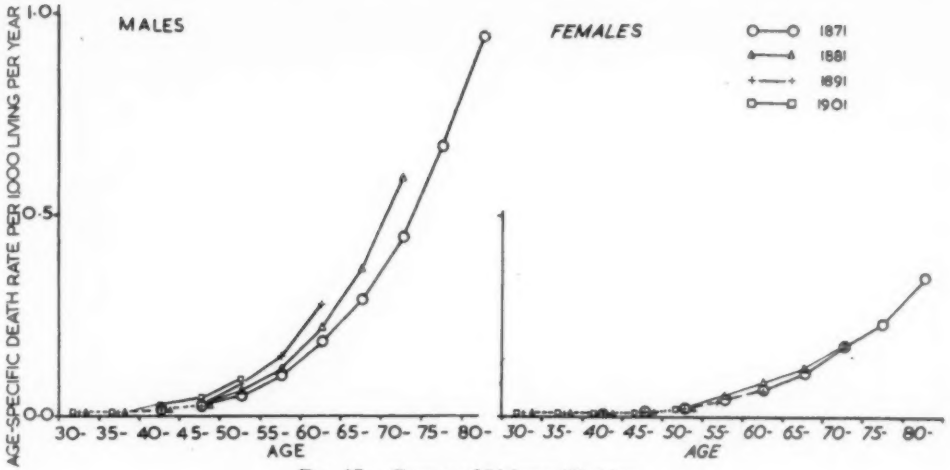


FIG. 17.—Cancer of Urinary Bladder.

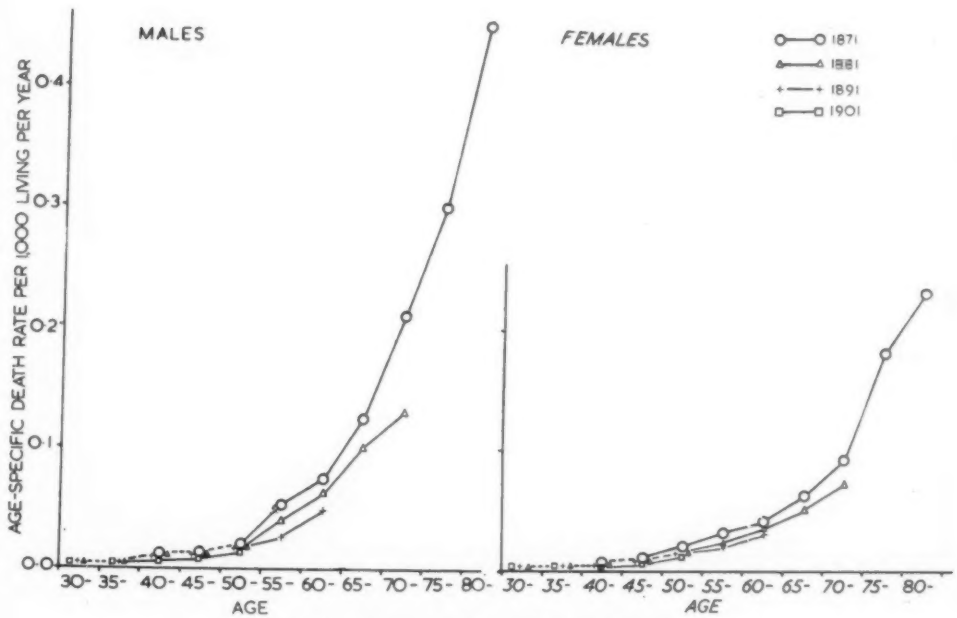


FIG. 18.—Cancer of Skin excluding Scrotum.

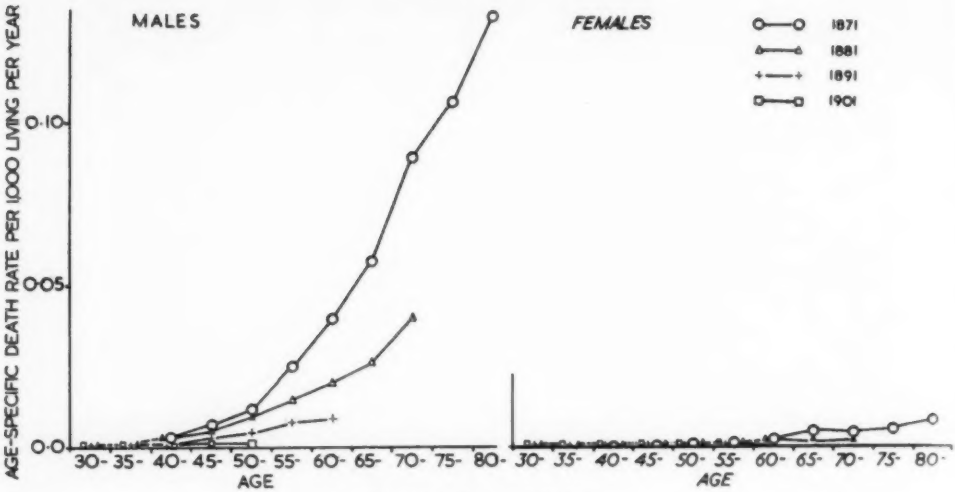


FIG. 19.—Cancer of Lips.

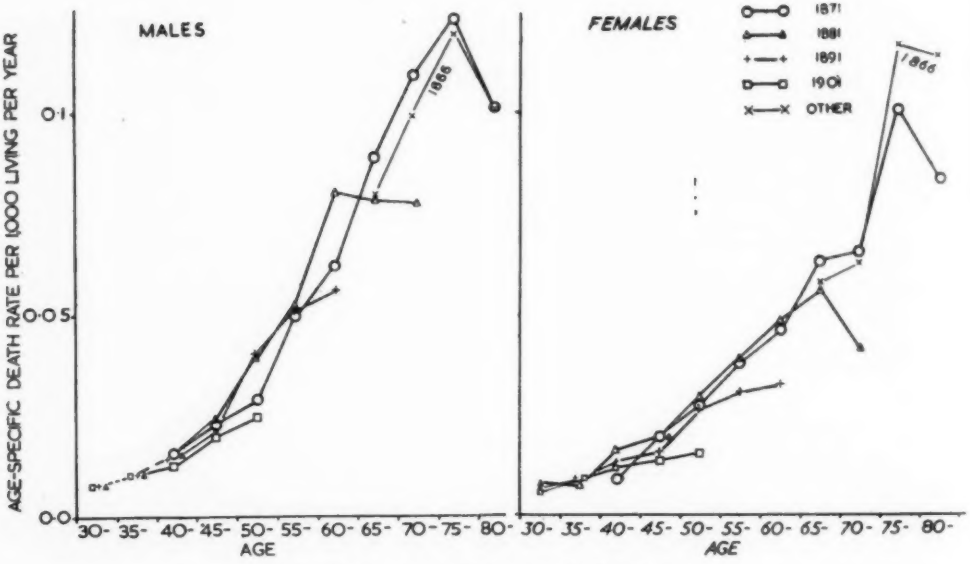


FIG. 20.—Cancer of Bones excluding Jaw.

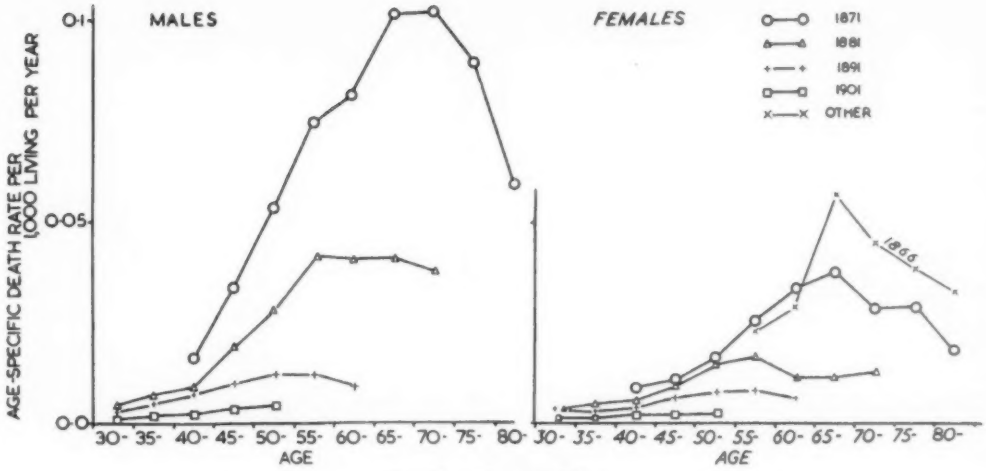


FIG. 21.—Cancer of Jaw.

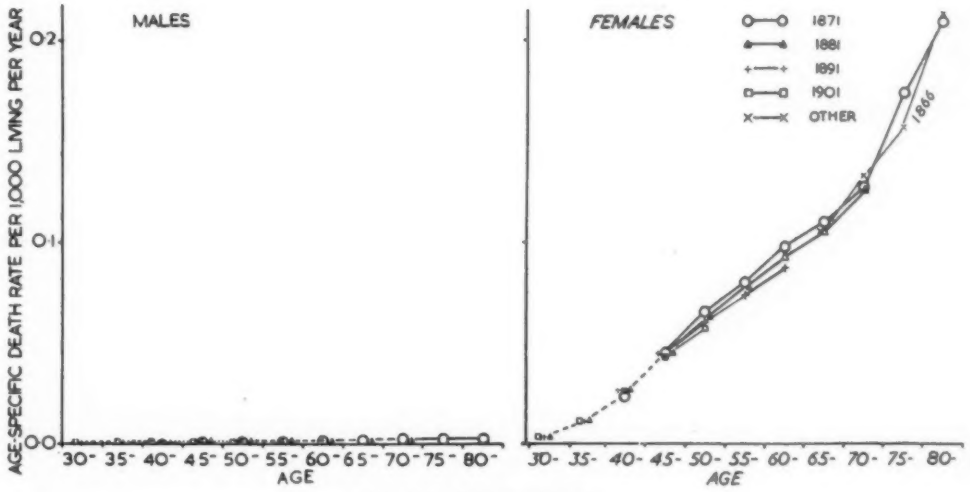


FIG. 22.—Cancer of Breast.

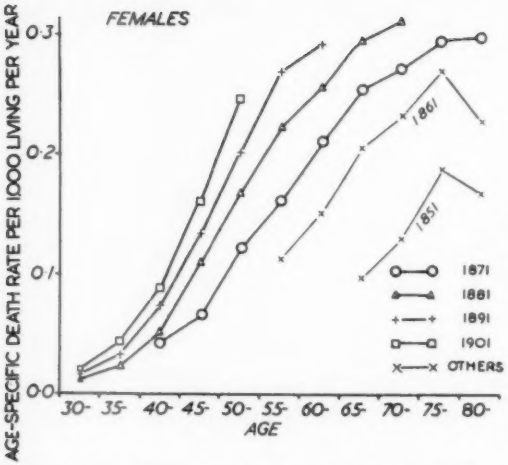


FIG. 23.—Cancer of Ovaries and Fallopian Tubes.

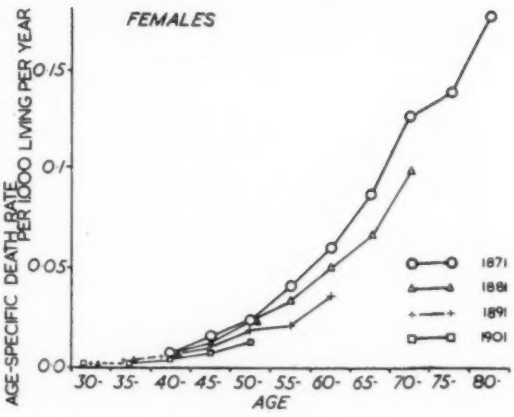


FIG. 24.—Cancer of Vulva and Vagina.

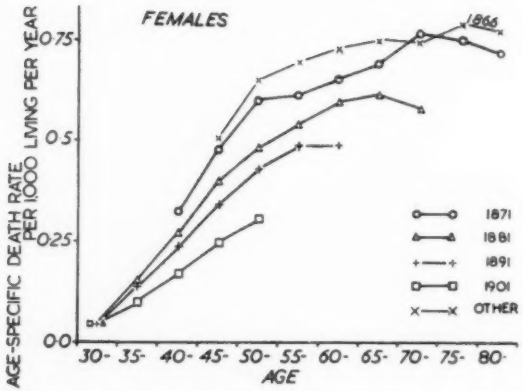


FIG. 25.—Cancer of Uterus.

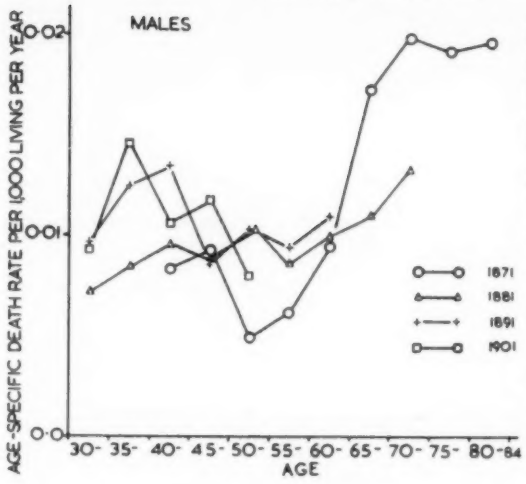


FIG. 26.—Cancer of Testes.

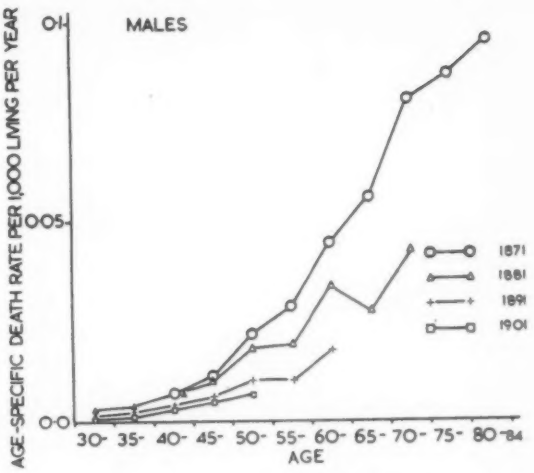


FIG. 27.—Cancer of Penis and Scrotum.

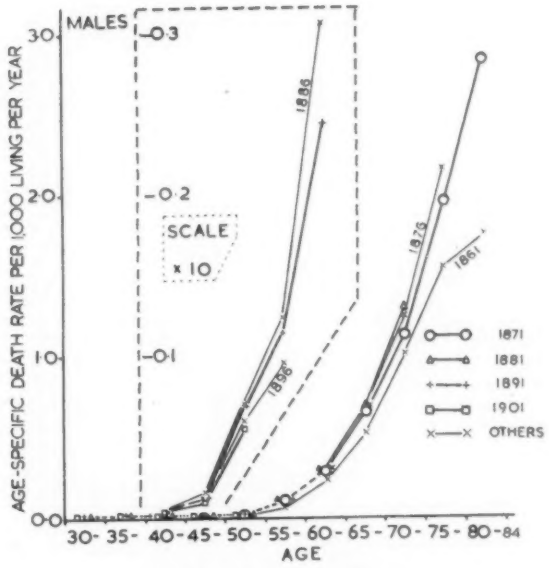


FIG. 28.—Cancer of Prostate.
(Inset: Tail of curve magnified ten times.)

Fig. 29 shows the aggregate of the sites not included amongst the 21 sites enumerated in the Tables.

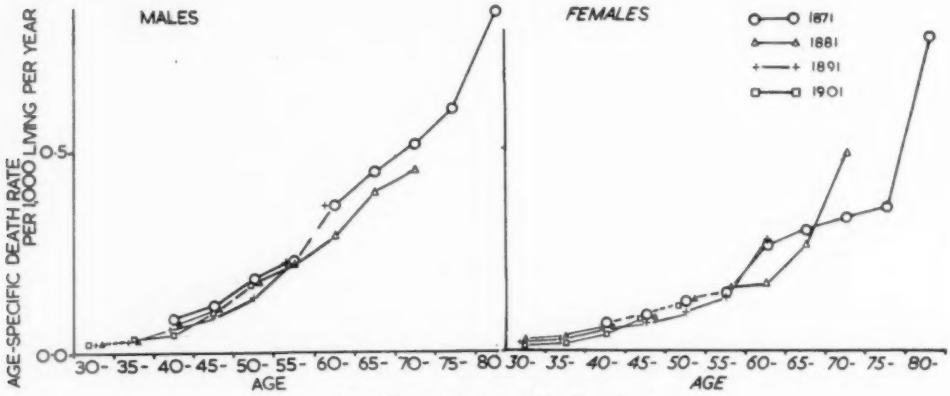
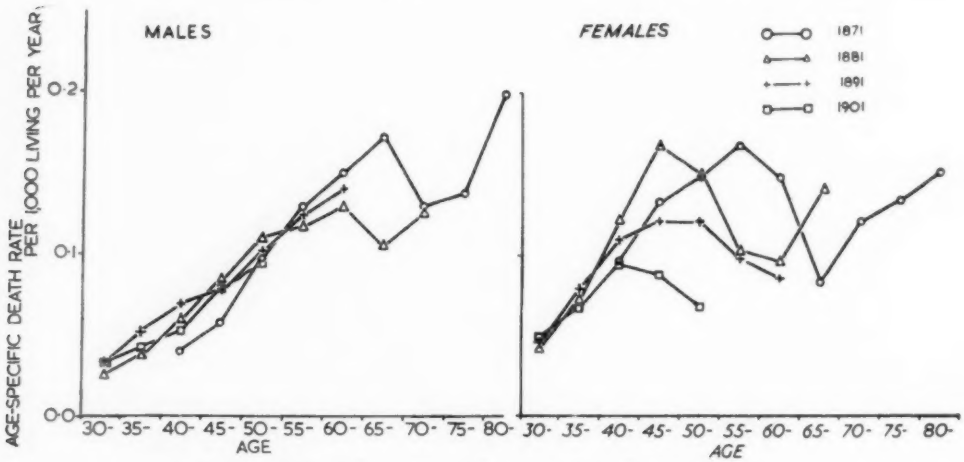


FIG. 29.—Cancer at All Other Sites.

Fig. 30, Benign Tumours and Tumours of Undetermined Nature, is put in for the sake of completeness.



FIG, 30.—Benign Tumours and Tumours of Undetermined Nature.

Fig. 31, Leukaemia, and Fig. 32, Hodgkin's Disease, (overleaf) are not included under the general heading of cancer because they are not universally accepted as such. In the Appendix Tables, the total of Cancer at All Sites, Benign Tumours and Tumours of Undetermined Nature, Leukaemia, and Hodgkin's Disease comprise the class called All Neoplasms.

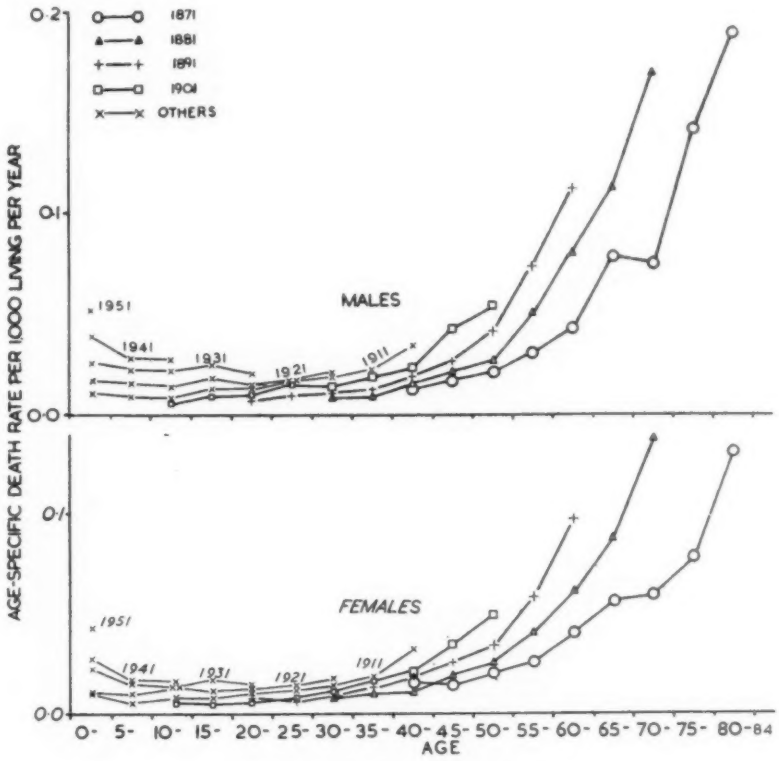


FIG. 31.—Leukaemia (All Types).

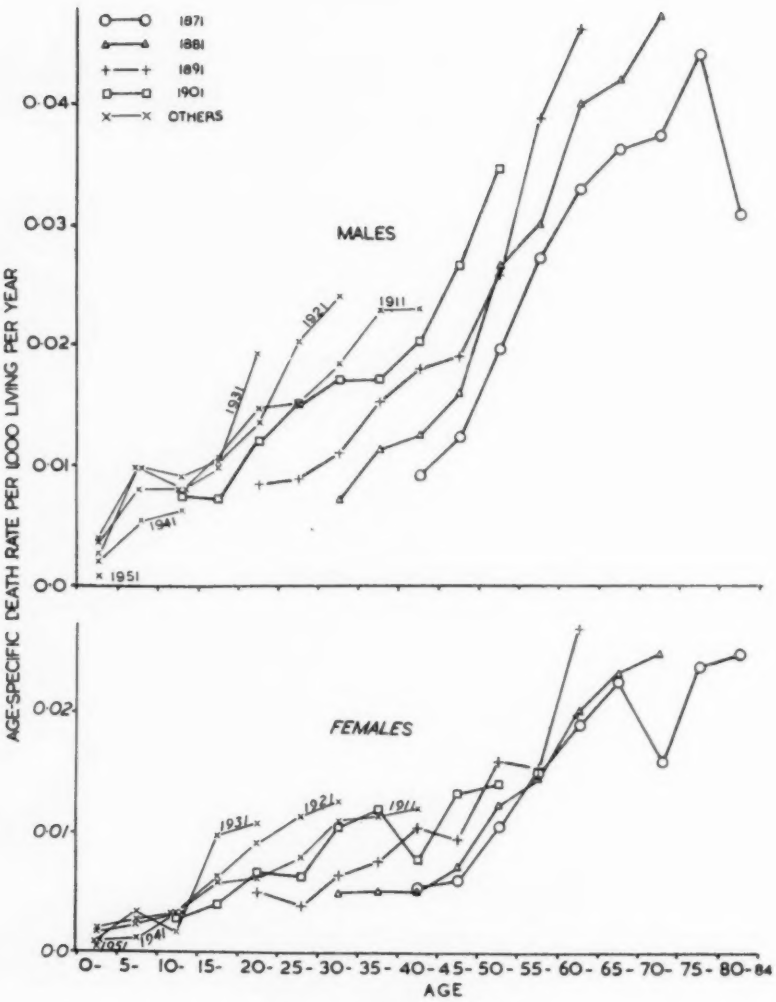


FIG. 32.—Hodgkin's Disease.

Now that the reader has become familiar with the graphical technique employed, it is unnecessary to comment on the picture for each individual site. It will suffice to group the sites together according to the behaviour of the death rates, and to comment on points of interest in the discussion.

(1) *Sites that show a Rising Rate*

(*a*) Male rise more pronounced.

Lungs (Fig. 5).
Kidneys and Adrenals (Fig. 16).
Urinary Bladder (Fig. 17) for males; female rate started to rise but is now steady.
Pancreas (Fig. 10).

(*b*) Rise about equal in both sexes.

Leukaemia (Fig. 31).
Hodgkin's Disease (Fig. 32).

(*c*) Sexual site.

Ovaries and Fallopian Tubes (Fig. 23).

(2) *Site that showed a Rising Rate but now shows a Falling Rate*

Prostate (Fig. 28).
Additional cohorts are used to show that a rising rate that persisted from at least the 1861 cohort until the 1881 cohort has now been replaced by a falling rate. The inset is the tail of the curve magnified ten times.

(3) *Sites that show No Definite Change*

Urinary Bladder (Fig. 17) for females.
Breast (Fig. 22) for males.
Testes (Fig. 26).

(4) *Sites that show a Falling Rate*

(*a*) Male fall more pronounced.

Oesophagus (Fig. 15).
Rectum (Fig. 8).
Tongue (Fig. 11).
Mouth and Tonsils (Fig. 12).
Larynx (Fig. 14).
Pharynx (Fig. 13) for males; female rate rising.
Jaw (Fig. 21).

(*b*) Female fall more pronounced.

Stomach (Fig. 6).
Intestines (Fig. 7).
Liver and Gall-Bladder (Fig. 9).
Skin, excluding Scrotum (Fig. 18).
Lips (Fig. 19).
Bones, excluding Jaw (Fig. 20).
Breast (Fig. 22) for females; male rate stationary.

(*c*) Sexual sites.

Penis and Scrotum (Fig. 27).
Uterus (Fig. 25).
Vulva and Vagina (Fig. 24).

(5) *Site that shows Anomalous Behaviour*

Pharynx (Fig. 13); male rate falling, female rate rising.

Discussion

The changes that are shown in the pictures are changes recorded in the death rates; as such they will reflect the effect of a complex of changing conditions that includes environment, diagnosis, treatment, and social habits. This paper makes no attempt to assess the influence of each of these factors in relation to the mortality changes shown at each site. A complete analysis of this sort may never be achieved, but even a partial one will require a study of the social history of these factors over the relevant period of time. The results of the study must then be correlated with cohort analysis to answer the question: "Could the changes of mortality shown have been due to the particular events that have given rise to some hypothesis of causation?" In this way the narrative properties of the method will be fully exploited.

There are, however, certain features in the graphs that can be discussed profitably even at this stage.

First, the changes in methods of death certification introduced in 1939 may cause discontinuities in the curves between the quinquennia 1936–40 and 1941–45. One such discontinuity occurs in the picture for female cancer of the breast (Fig. 22). That the kink is in fact an artefact is shown by using part of an additional cohort line for 1866. The kink that was seen at age 70–74 in the 1871 cohort now moves to age 75–79, corresponding to the year 1940.

Secondly, a peak that moves to different ages in successive cohorts, such as that shown by the Bones, excluding Jaw (Fig. 20) and the Jaw (Fig. 21), could reflect a change in either aetiology or therapy that took place at a sharply-defined period of time. In the case of aetiology this time might occur many years before the change shown in the picture. It is interesting to speculate whether a revolution in anaesthesia, surgical technique, and post-operative treatment between about 1936 and 1945 could have produced a dramatic rise in the cure rate, and hence a fall in the death rate, at these sites. Even if these factors could be invoked to explain the shift in the peak, the use of additional cohorts reveals that there was previously a stationary

peak at an advanced age. A peak at a very advanced age might be of diagnostic origin because of an unwillingness to subject an elderly patient to distressing diagnostic procedures.

Thirdly, when rates are changing, some consideration must be given to the relative changes shown by each sex. Thus, it might be reasonable to attribute the changes in the death rate for, say, the Liver and Gall-Bladder (Fig. 9), which shows a decrease about equal in both sexes, to better allocation of the disease to a primary site, and those in the death rate for Leukaemia (Fig. 31) and Hodgkin's Disease (Fig. 32), which show an increase about equal in both sexes, to improvements in diagnosis; but would it be reasonable to use the same argument in the case of the Lungs (Fig. 5), Urinary Bladder (Fig. 17) and Kidneys and Adrenals (Fig. 16), all of which show a predominantly male rise? A detailed knowledge of how the diagnosis has been made and recorded for each sex at different times is needed to answer this question.

In certain sites, such as the Oesophagus (Fig. 15), Rectum (Fig. 8), Tongue (Fig. 11), Mouth and Tonsils (Fig. 12), and Larynx (Fig. 14), there is a disproportionate fall in the male death rate. Can this be accounted for by a difference in the results of therapy or in the availability of therapy, or should some explanation involving a differentially changed environment be invoked?

Where a substantial fall in death rates for both sexes is shown, do current views of the results of therapy support a contention that the fall is a reflection of therapeutic triumphs or should the reason be sought in an environmental change common to both sexes?

One of the purposes of presenting these studies is to provide part of the basic information necessary to make renewed attempts to answer these complex questions, and to allow other pertinent questions to be formulated. Another purpose is to answer the three questions posed in the introductory paragraphs.

Question 1.—Is the lethal force with which cancer affects the community increasing, and if so what forms of cancer contribute to this increase?

Answer.—In the male, Yes; but if cancer of the Lungs is excluded, No. (Reasons for considering the problem with and without exclusion are given above). In the female, No.

Question 2.—Is there, generally speaking, a "cancer-age", or does the force of mortality due to cancer at different sites increase throughout the life-span?

Answer.—In general, no "cancer-age" is found, and the force of mortality increases throughout the life-span. Exceptions to this are cancer of the Uterus and possibly cancer of the Bones, Jaw, and Tongue, where the rates show a decline at advanced ages. Such a late decline may be of diagnostic origin. The curves for cancer of the Testes are too confused to allow a conclusion to be drawn.

Question 3.—Is there a consistent sex-difference in the force of mortality due to cancer at the different sites common to both sexes, and if so how great is this difference?

Answer.—In thirteen of the eighteen sites common to both sexes that were studied, the male showed an equal or higher mortality rate at all the ages analysed. (The lowest age was 30 in three of the cohorts and 40 in the other).

At four sites, Larynx, Oesophagus, Intestines, and Liver and Gall-Bladder, the female rate was initially (age 30–59) slightly higher. A male preponderance was established before the age of 45 for the Larynx and Oesophagus, and before the age of 60 for the Intestines and Liver and Gall-Bladder and continued thereafter.

In the remaining site, the Breast, the female had a much higher rate at all ages, but the male organ is vestigial and the comparison unfair.

The sex ratio at ages above 60 varies from about 1·2 : 1 to about 10 : 1 for the seventeen sites showing a male preponderance, but the ratio may change from age group to age group and from cohort to cohort.

An unwary reader might be tempted to add together the cancer death rates for males and females and to assert that the constancy shown by this compound figure supports the hypothesis of Cramer (19 6) that the cancer death rate in a community remains constant, and that an alteration of the rate at one site is compensated by a concomitant variation at another site.

From a statistical point of view a concept of this sort can be construed in two ways:

The first is to say that a given individual has a certain tendency to develop cancer, and that if cancer does not appear in one site there is a greater liability for it to appear in another. Although I am unaware of any valid grounds for holding this view, it does not, *a priori*, appear biologically absurd.

The second is to say that if Person A alters his habits or environment in such a way that he changes the likelihood of his developing cancer, this will affect, in the opposite way, the chances of a Person B developing cancer, the sum of the two likelihoods remaining constant. This is unlikely to commend itself to many scientists as a biological proposition, but it is the construction that must be invoked if the male and female rates are added together and used as evidence of an unchanging cancer mortality.

SUMMARY

The death rates for cancer at 21 sites for both males and females in England and Wales are shown in graphical form, analysed by cohort analysis covering the years 1911–1954.

The graphs are intended primarily as an atlas of cancer mortality trends, to serve as a background for further studies of the reasons for these trends.

The death-rate from Cancer at All Sites is rising in males and falling in females. If cancer of the Lungs is excluded, the trend is to fall in both sexes, and the mortality rates at the age of 50–54 affecting persons born around 1901 are 70 per cent. (males) and 80 per cent. (females) of the rates that affected persons born 30 years earlier (*i.e.* around 1871) at the age of 50–54.

The Lungs, Pancreas, Urinary Bladder in males and the Kidneys and Adrenals, Ovaries and Fallopian Tubes, and Pharynx in females are the only sites of cancer to show a rising death rate. By far the greatest rise is shown by cancer of the Lungs in males. Some sites show a proportionately much greater improvement in the death rate for males than for females.

In general, no "cancer-age" is found, the death rates continuing to rise throughout life. Exceptions to this are cancer of the Uterus, and a very late effect, possibly of diagnostic origin, for cancer of the Bones, the Jaw, and the Tongue.

The male death rates at ages over 60 are always higher than the female death rates for the sites of cancer common to both sexes that were studied. (The Breast forms an exception to this rule, but the male organ is vestigial and the comparison unfair.) Between the ages of 30 and 45 for the sites Larynx and Oesophagus and between the ages 30 and 60 for the sites Intestines and Liver and Gall-Bladder, the female rates exceed the male rates. After these ages a male preponderance is established.

The ratio of male to female mortality may change from site to site, age group to age group, and cohort to cohort.

Thanks are due to Miss Joan Pearson (now Mrs. G. Bennette) and to Mrs. W. A. Ward for assembling data and computing rates, and to the Registrar-General's staff for supplying some unpublished data.

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APPENDIX

DETAILS OF THE SOURCES OF INFORMATION FOR, AND THE METHODS OF, CALCULATING THE RATES

From 1911 until 1949 the successive volumes of the Registrar-General's "Reports" and "Reviews" contained a Table showing the number of deaths from cancer occurring in England and Wales in each year. These cancer deaths were subdivided according to sex, age, and site, and included non-civilians. The Table was always in the "Text" part of the Report or Review, and was variously called "Sites of Fatal Cancer" (1911–1930), "Forms of Fatal Cancer at Each Site" (1931–1938), and then "Cancer of Different Forms and Sites" (1939–49). In 1950 this Table was discontinued, but subsequently Table 17 of the "Tables (Medical) Section" gave a fairly detailed breakdown of cancer by site. The

Registrar-General's staff kindly supplied the additional information necessary to reconstruct the remaining relevant sections of the discontinued Table.

Of the 26 sites listed for each sex in 1911, 21 could be followed throughout the ensuing 40 years by careful scrutiny of the Tables and the definitions given in the various manuals based on the "International List of Causes of Death" (H.M.S.O., Various Years), which was revised in 1909, 1920, 1929, 1938, and 1948. In addition to giving the data by sites, the Tables gave figures for Cancer at All Sites. The difference between these figures and the sum of the 21 sites selected for the analysis gave a category "Cancer at All Other Sites" (Fig. 29), which varied from about 5 per cent. to about 10 per cent. of the total according to age and date. In other words the breakdown by sites covered more than 90 per cent. of the total cancer mortality.

Leukaemia, Hodgkin's Disease, and Benign Tumours and Tumours of Undetermined Nature were listed under a variety of titles in the Tables relating to "Causes of Death at Different Age Periods" that appeared in the "Tables Section" of the "Reports" and "Reviews". During some of the war years these Tables excluded non-civilians, but their deaths were listed in a special table, and were added to the totals.

Hodgkin's Disease appeared under the following headings and code numbers:

Lymphadenoma 53b (1911–1920).
Lymphadenoma (Hodgkin's Disease) 65b (1921–30).
Aleukaemia (Lymphadenoma) 72b (1931–38).
Pernicious Lymphogranulomatosis 44b (1939–49).
Hodgkin's Disease 201 (1950 and subsequently).

Leukaemia was listed under the following headings and code numbers:

Leucocythaemia (Leuchaemia) 53a (1911–1920).
Leukaemia 65a (1921–30).
Leukaemia 72a (1931–1938).
Leukaemia and Aleukaemia 74 (1939–1949).
Leukaemia and Aleukaemia 204 (1950 and subsequently).

Benign Tumours and Tumours of Undetermined Nature were listed under this title or one very like it. Tumours of the Central Nervous System and Benign Tumours of the Uterus and Adnexa were added to these figures in those years where they were not already included.

The age breakdown was fairly detailed but inconsistent. The "Cancer by Site" Tables ran 0–, 5–, 15–, 25–, 35–, 40–, 45–, and thereafter in quinary groups until the ultimate group 85 and over. The "Causes of Death at Different Age Periods" ran in quinary groups from birth until an ultimate group of 80 and over in some years or 85 and over in others.

The mid-year population estimates were given in the same series of "Reports" and "Reviews". For some years a Revised Estimate appeared later, when Census returns had made this possible. These revised tables were used when available. Populations designated as "including non-civilians" were used until 1949. Thereafter the populations called "Home Population" were used as being the most appropriate. All these population Tables gave a uniform quinary age breakdown up to an ultimate group of 85 and over.

The data (populations and deaths) were combined into quinquennial groups, 1911–15, 1916–20, and so on. Since the 1955 figures had not become available at the time of writing, the four years 1951, 2, 3, and 4 were used to represent the quinquennium.

The age-specific death rates were calculated by dividing the total number of deaths in each quinary age group in each quinquennium by the sum of the mid-year populations for the appropriate age group and quinquennium. Since the mid-year estimate of the population was a very close approximation to the number of years of life-experience at the stated age in the year, the resulting death rates were the average number of deaths occurring per 1,000 years of life-experience in a specified 5-year age-group during a specified 5-year period.

Correction factors were computed in an age-specific manner by dividing the entries in Table 21 (p. 113) of the "Review" of the Registrar-General (1942) by the corresponding entries in Appendix B.1 (p. 301) of the same volume. Reciprocals of this ratio were also calculated. This gave two age-specific factors that could convert pre-1939 death rates to post-1939 ones or *vice versa*. In these studies all the rates subsequent to 1939 were converted to pre-1939 rates. The published data were insufficient to allow correction factors to be calculated for all sites. The sites where a factor was obtained are indicated in Appendix Table I.

Rates for the age group 30–34 were in many instances obtained by graphical interpolation because of the change in age-grouping in the published data. Appendix Table I shows where this was done.

APPENDIX TABLE I

AGE-SPECIFIC DEATH RATES AT AGES 30–84, IN QUINARY GROUPS, FOR THE FOUR MAIN COHORTS BORN AROUND 1871, 1881, 1891, AND 1901

All rates are expressed as rates per 1,000 living per year. *Female* rates are in italic figures. Male rates are not.

The symbol • indicates that the rates for this age and site were found by curvilinear graphical interpolation from the inconsistently grouped data.

The symbol (C) indicates that a correction factor for the 1939 change in death certification procedure had been applied at this site.

Fig. No.	Cause of Death	Cohort born	Age 30–	35–	40–	45–	50–	55–	60–	65–	70–	75–	80–84
2	All Causes (General Mortality)	1871	—	—	9·176	11·894	13·425	19·673	28·783	45·814	64·278	101·751	165·668
			—	—	*7·196*	*8·867*	*10·353*	*14·331*	*21·074*	*32·821*	*46·778*	*76·904*	*127·025*
		1881	5·584	7·800	7·291	9·836	13·154	20·101	28·227	41·789	68·143	—	—
			4·452	*5·952*	*5·504*	*7·007*	*9·361*	*12·869*	*17·190*	*26·000*	*43·191*	—	—
		1891	4·298	5·242	6·221	8·605	11·860	17·423	28·542	—	—	—	—
			3·755	*4·168*	*4·796*	*6·122*	*7·598*	*9·963*	*15·024*	—	—	—	—
		1901	3·475	4·234	5·035	6·703	10·552	—	—	—	—	—	—
			3·212	*3·373*	*3·703*	*4·473*	*6·052*	—	—	—	—	—	—
—	All Neoplasms (C)	1871	—	—	0·667	1·262	2·334	3·896	6·150	8·976	11·979	15·626	18·869
			—	—	*1·212*	*1·964*	*2·849*	*3·751*	*5·103*	*6·571*	*8·319*	*10·853*	*13·354*
		1881	0·189	0·317	0·634	1·240	2·276	3·837	6·121	9·145	12·826	—	—
			0·286	*0·612*	*1·150*	*1·885*	*2·697*	*3·581*	*4·735*	*6·155*	*7·794·*	—	—
		1891	0·198	0·355	0·675	1·292	2·369	4·296	6·980	—	—	—	—
			0·283	*0·614*	*1·112*	*1·789*	*2·558*	*3·371*	*4·372*	—	—	—	—
		1901	0·218	0·402	0·772	1·579	2·899	—	—	—	—	—	—
			0·292	*0·587*	*1·029*	*1·671*	*2·308*	—	—	—	—	—	—
3	Cancer at All Sites (C)	1871	—	—	0·605	1·175	2·198	3·709	5·925	8·786	11·739	15·308	18·457
			—	—	*1·090*	*1·813*	*2·670*	*3·564*	*4·898*	*6·408*	*8·122*	*10·615*	*13·043*
		1881	0·147	0·259	0·547	1·118	2·111	3·643	5·871	8·886	12·483	—	—
			0·232	*0·524*	*1·023*	*1·694*	*2·509*	*3·417*	*4·550*	*5·950*	*7·490*	—	—
		1891	0·145	0·276	0·570	1·173	2·200	4·055	6·666	—	—	—	—
			0·224	*0·517*	*0·976*	*1·633*	*2·391*	*3·199*	*4·161*	—	—	—	—
		1901	0·155	0·322	0·677	1·432	2·690	—	—	—	—	—	—
			0·224	*0·488*	*0·918*	*1·534*	*2·187*	—	—	—	—	—	—
4	Cancer at All Sites except Lungs (C)	1871	—	—	0·588	1·150	2·132	3·561	5·565	8·150	10·968	14·212	17·096
			—	—	*1·079*	*1·799*	*2·644*	*3·506*	*4·780*	*6·229*	*7·891*	*10·300*	*12·647*
		1881	0·140	0·248	0·520	1·042	1·855	3·057	4·840	7·066	9·925	—	—
			0·229	*0·519*	*1·011*	*1·671*	*2·454*	*3·310*	*4·276*	*5·642*	*7·105*	—	—
		1891	0·137	0·254	0·483	0·899	1·597	2·676	4·185	—	—	—	—
			0·221	*0·508*	*0·951*	*1·584*	*2·297*	*3·028*	*3·875*	—	—	—	—
		1901	0·135	0·254	0·484	0·882	1·459	—	—	—	—	—	—
			0·217 •	*0·472*	*0·882*	*1·460*	*2·052*	—	—	—	—	—	—
5	Cancer of Lungs (C)	1871	—	—	0·017	0·025	0·066	0·148	0·360	0·636	0·771	1·096	1·361
			—	—	*0·011*	*0·014*	*0·026*	*0·058*	*0·118*	*0·179*	*0·231*	*0·315*	*0·396*
		1881	0·007	0·011	0·027	0·076	0·256	0·586	1·031	1·820	2·558	—	—
			0·003	*0·005*	*0·012*	*0·023*	*0·055*	*0·107*	*0·174*	*0·308*	*0·385*	—	—
		1891	0·008	0·022	0·087	0·274	0·603	1·379	2·481	—	—	—	—
			0·003	*0·009*	*0·025*	*0·049*	*0·094*	*0·171*	*0·285*	—	—	—	—
		1901	0·020	0·068	0·193	0·550	1·231	—	—	—	—	—	—
			0·007 •	*0·016*	*0·036*	*0·074*	*0·135*	—	—	—	—	—	—
6	Cancer of Stomach (C)	1871	—	—	0·139	0·272	0·515	0·866	1·354	1·958	2·405	2·946	3·178
			—	—	*0·102*	*0·182*	*0·342*	*0·538*	*0·845*	*1·253*	*1·595*	*2·174*	*2·704*
		1881	0·024	0·064	0·156	0·335	0·544	0·878	1·310	1·847	2·431	—	—
			0·021	*0·051*	*0·099*	*0·187*	*0·313*	*0·471*	*0·736*	*1·055*	*1·365*	—	—
		1891	0·029	0·074	0·161	0·309	0·516	0·836	1·274	—	—	—	—
			0·018	*0·049*	*0·095*	*0·166*	*0·254*	*0·381*	*0·538*	—	—	—	—
		1901	0·030	0·077	0·155	0·279	0·478	—	—	—	—	—	—
			0·023 •	*0·050*	*0·080*	*0·132*	*0·206*	—	—	—	—	—	—
7	Cancer of Intestines (C)	1871	—	—	0·058	0·111	0·204	0·421	0·655	1·170	1·743	2·372	2·680
			—	—	*0·072*	*0·138*	*0·252*	*0·412*	*0·596*	*0·968*	*1·470*	*2·049*	*2·437*
		1881	0·019	0·030	0·062	0·114	0·203	0·391	0·696	1·009	1·421	—	—
			0·021	*0·034*	*0·071*	*0·140*	*0·224*	*0·371*	*0·609*	*0·848*	*1·096*	—	—
		1891	0·019	0·033	0·058	0·110	0·201	0·337	0·472	—	—	—	—
			0·020	*0·036*	*0·071*	*0·144*	*0·234*	*0·343*	*0·470*	—	—	—	—
		1901	0·017	0·031	0·079	0·114	0·162	—	—	—	—	—	—
			0·022 •	*0·045*	*0·084*	*0·136*	*0·186*	—	—	—	—	—	—

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APPENDIX TABLE I (*contin.*)

Fig. No.	Cause of Death	Cohort born	Age 30–	35–	40–	45–	50–	55–	60–	65–	70–	75–	80–84
8	Cancer of Rectum (C)	1871	—	—	0·056	0·101	0·208	0·375	0·659	1·027	1·487	1·875	2·086
			—	—	*0·048*	*0·084*	*0·135*	*0·206*	*0·312*	*0·418*	*0·618*	*0·832*	*0·994*
		1881	0·016	0·026	0·045	0·092	0·187	0·350	0·605	0·906	1·149	—	—
			0·016	*0·027*	*0·049*	*0·072*	*0·110*	*0·189*	*0·290*	*0·396*	*0·497*	—	—
		1891	0·015	0·021	0·046	0·082	0·169	0·296	0·415	—	—	—	—
			0·015	*0·021*	*0·042*	*0·071*	*0·122*	*0·179*	*0·242*	—	—	—	—
		1901	0·016	0·023	0·052	0·082	0·130	—	—	—	—	—	—
			0·012	*0·025*	*0·042*	*0·070*	*0·100*	—	—	—	—	—	—
			*										
9	Cancer of Liver and Gall-Bladder (C)	1871	—	—	0·050	0·099	0·152	0·201	0·297	0·364	0·445	0·449	0·512
			—	—	*0·066*	*0·119*	*0·164*	*0·216*	*0·290*	*0·347*	*0·448*	*0·458*	*0·477*
		1881	0·009	0·022	0·036	0·058	0·090	0·142	0·214	0·257	0·320	—	—
			0·011	*0·026*	*0·040*	*0·063*	*0·094*	*0·144*	*0·196*	*0·241*	*0·284*	—	—
		1891	0·008	0·010	0·024	0·039	0·072	0·105	0·162	—	—	—	—
			0·008	*0·013*	*0·025*	*0·040*	*0·069*	*0·088*	*0·132*	—	—	—	—
		1901	0·005	0·009	0·019	0·037	0·055	—	—	—	—	—	—
			0·004	*0·010*	*0·019*	*0·030*	*0·050*	—	—	—	—	—	—
			*										
10	Cancer of Pancreas (C)	1871	—	—	0·015	0·027	0·059	0·120	0·185	0·293	0·388	0·574	0·736
			—	—	*0·010*	*0·019*	*0·043*	*0·087*	*0·145*	*0·214*	*0·270*	*0·398*	*0·523*
		1881	0·004	0·007	0·018	0·043	0·084	0·131	0·201	0·333	0·518	—	—
			0·002	*0·005*	*0·012*	*0·026*	*0·051*	*0·093*	*0·139*	*0·226*	*0·322*	—	—
		1891	0·005	0·009	0·020	0·043	0·087	0·148	0·261	—	—	—	—
			0·003	*0·007*	*0·015*	*0·029*	*0·050*	*0·098*	*0·143*	—	—	—	—
		1901	0·004	0·007	0·021	0·049	0·091	—	—	—	—	—	—
			0·003	*0·005*	*0·013*	*0·025*	*0·054*	—	—	—	—	—	—
			*										
11	Cancer of Tongue (C)	1871	—	—	0·036	0·080	0·142	0·205	0·269	0·297	0·355	0·343	0·339
			—	—	*0·004*	*0·009*	*0·008*	*0·016*	*0·023*	*0·027*	*0·039*	*0·053*	*0·059*
		1881	0·002	0·005	0·018	0·040	0·062	0·088	0·128	0·140	0·171	—	—
			0·001	*0·002*	*0·003*	*0·006*	*0·008*	*0·013*	*0·017*	*0·024*	*0·035*	—	—
		1891	0·002	0·003	0·005	0·013	0·023	0·035	0·046	—	—	—	—
			0·001	*0·002*	*0·002*	*0·004*	*0·009*	*0·009*	*0·014*	—	—	—	—
		1901	0·001	0·001	0·003	0·005	0·009	—	—	—	—	—	—
			0·001	*0·002*	*0·002*	*0·004*	*0·006*	—	—	—	—	—	—
			*										
12	Cancer of Mouth and Tonsils	1871	—	—	0·015	0·036	0·078	0·137	0·145	0·173	0·178	0·196	0·345
			—	—	*0·002*	*0·007*	*0·008*	*0·012*	*0·013*	*0·019*	*0·019*	*0·030*	*0·058*
		1881	0·002	0·003	0·010	0·026	0·034	0·048	0·064	0·083	0·160	—	—
			0·001	*0·002*	*0·002*	*0·006*	*0·007*	*0·008*	*0·009*	*0·012*	*0·024*	—	—
		1891	0·001	0·004	0·004	0·009	0·013	0·020	0·040	—	—	—	—
			0·001	*0·001*	*0·003*	*0·002*	*0·003*	*0·008*	*0·013*	—	—	—	—
		1901	0·002	0·002	0·002	0·005	0·014	—	—	—	—	—	—
			0·000	*0·002*	*0·001*	*0·002*	*0·005*	—	—	—	—	—	—
			*										
13	Cancer of Pharynx	1871	—	—	0·007	0·020	0·032	0·059	0·095	0·124	0·137	0·159	0·196
			—	—	*0·005*	*0·009*	*0·012*	*0·010*	*0·016*	*0·023*	*0·023*	*0·030*	*0·042*
		1881	0·001	0·002	0·005	0·014	0·028	0·038	0·062	0·078	0·124	—	—
			0·001	*0·003*	*0·006*	*0·007*	*0·010*	*0·016*	*0·013*	*0·024*	*0·043*	—	—
		1891	0·001	0·002	0·003	0·010	0·018	0·025	0·049	—	—	—	—
			0·001	*0·002*	*0·003*	*0·007*	*0·010*	*0·016*	*0·030*	—	—	—	—
		1901	0·001	0·002	0·006	0·009	0·016	—	—	—	—	—	—
			0·001	*0·002*	*0·003*	*0·007*	*0·019*	—	—	—	—	—	—
			*										
14	Cancer of Larynx (C)	1871	—	—	0·016	0·044	0·097	0·158	0·229	0·295	0·351	0·344	0·396
			—	—	*0·010*	*0·023*	*0·026*	*0·039*	*0·037*	*0·047*	*0·048*	*0·062*	*0·044*
		1881	0·001	0·003	0·015	0·035	0·073	0·117	0·163	0·207	0·248	—	—
			0·003	*0·006*	*0·013*	*0·020*	*0·029*	*0·034*	*0·039*	*0·039*	*0·036*	—	—
		1891	0·001	0·004	0·010	0·024	0·043	0·083	0·116	—	—	—	—
			0·002	*0·005*	*0·009*	*0·014*	*0·027*	*0·027*	*0·023*	—	—	—	—
		1901	0·001	0·003	0·009	0·024	0·036	—	—	—	—	—	—
			0·002	*0·005*	*0·007*	*0·012*	*0·013*	—	—	—	—	—	—
			*										
15	Cancer of Oesophagus (C)	1871	—	—	0·035	0·091	0·197	0·297	0·457	0·560	0·621	0·789	0·857
			—	—	*0·025*	*0·038*	*0·062*	*0·072*	*0·114*	*0·144*	*0·168*	*0·261*	*0·338*
		1881	0·002	0·005	0·018	0·053	0·107	0·193	0·260	0·370	0·475	—	—
			0·005	*0·011*	*0·020*	*0·030*	*0·055*	*0·070*	*0·099*	*0·135*	*0·196*	—	—
		1891	0·002	0·004	0·012	0·026	0·062	0·114	0·185	—	—	—	—
			0·003	*0·008*	*0·015*	*0·026*	*0·038*	*0·063*	*0·085*	—	—	—	—
		1901	0·002	0·006	0·015	0·027	0·054	—	—	—	—	—	—
			0·002	*0·005*	*0·010*	*0·018*	*0·032*	—	—	—	—	—	—
			*										

—*contin.*

APPENDIX TABLE I (*contin.*)

Fig. No.	Cause of Death	Cohort born	Age 30–	35–	40–	45–	50–	55–	60–	65–	70–	75–	80–84
16	Cancer of Kidneys and Adrenals	1871	—	—	0·008	0·015	0·027	0·051	0·073	0·086	0·081	0·111	0·130
			—	—	*0·007*	*0·010*	*0·018*	*0·027*	*0·045*	*0·056*	*0·059*	*0·085*	*0·115*
		1881	0·003	0·005	0·010	0·019	0·035	0·049	0·073	0·106	0·147	—	—
			0·002	*0·004*	*0·008*	*0·011*	*0·024*	*0·031*	*0·039*	*0·057*	*0·085*	—	—
		1891	0·003	0·007	0·015	0·025	0·038	0·069	0·112	—	—	—	—
			0·003	*0·003*	*0·008*	*0·014*	*0·020*	*0·030*	*0·048*	—	—	—	—
		1901	0·005	0·008	0·013	0·029	0·054	—	—	—	—	—	—
			0·003 •	*0·004*	*0·008*	*0·013*	*0·021*	—	—	—	—	—	—
17	Cancer of Urinary Bladder	1871	—	—	0·013	0·024	0·048	0·100	0·183	0·285	0·443	0·665	0·941
			—	—	*0·004*	*0·010*	*0·022*	*0·036*	*0·059*	*0·106*	*0·166*	*0·239*	*0·346*
		1881	0·002	0·005	0·012	0·021	0·062	0·114	0·220	0·355	0·586	—	—
			0·001	*0·004*	*0·006*	*0·014*	*0·018*	*0·040*	*0·082*	*0·130*	*0·167*	—	—
		1891	0·001	0·006	0·013	0·034	0·079	0·143	0·272	—	—	—	—
			0·001	*0·002*	*0·006*	*0·010*	*0·028*	*0·041*	*0·064*	—	—	—	—
		1901	0·002	0·005	0·012	0·039	0·088	—	—	—	—	—	—
			0·001 •	*0·003*	*0·007*	*0·014*	*0·025*	—	—	—	—	—	—
18	Cancer of Skin (excluding Scrotum) (C)	1871	—	—	0·020	0·021	0·030	0·055	0·075	0·123	0·216	0·301	0·477
			—	—	*0·008*	*0·013*	*0·022*	*0·031*	*0·042*	*0·060*	*0·090*	*0·178*	*0·231*
		1881	0·004	0·006	0·009	0·015	0·026	0·041	0·063	0·100	0·133	—	—
			0·002	*0·004*	*0·010*	*0·011*	*0·018*	*0·025*	*0·032*	*0·052*	*0·072*	—	—
		1891	0·004	0·006	0·007	0·016	0·020	0·028	0·046	—	—	—	—
			0·003	*0·006*	*0·006*	*0·013*	*0·016*	*0·021*	*0·029*	—	—	—	—
		1901	0·004	0·004	0·007	0·015	0·020	—	—	—	—	—	—
			0·003 •	*0·005*	*0·009*	*0·011*	*0·012*	—	—	—	—	—	—
19	Cancer of Lips (C)	1871	—	—	0·003	0·007	0·012	0·026	0·040	0·056	0·089	0·109	0·135
			—	—	*0·000*	*0·001*	*0·001*	*0·001*	*0·002*	*0·004*	*0·004*	*0·005*	*0·008*
		1881	0·000	0·001	0·002	0·005	0·010	0·016	0·021	0·027	0·037	—	—
			0·000	*0·000*	*0·000*	*0·001*	*0·001*	*0·001*	*0·002*	*0·002*	*0·004*	—	—
		1891	0·000	0·001	0·002	0·003	0·004	0·006	0·009	—	—	—	—
			0·000	*0·000*	*0·000*	*0·000*	*0·000*	*0·001*	*0·001*	—	—	—	—
		1901	0·000	0·000	0·001	0·002	0·001	—	—	—	—	—	—
			0·000 •	*0·000*	*0·000*	*0·000*	*0·000*	—	—	—	—	—	—
20	Cancer of Bones (excluding Jaw) (C)	1871	—	—	0·016	0·023	0·029	0·050	0·063	0·090	0·110	0·129	0·102
			—	—	0·009	*0·020*	*0·028*	*0·038*	*0·045*	*0·063*	*0·065*	*0·105*	*0·083*
		1881	0·009	0·011	0·014	0·024	0·039	0·052	0·081	0·079	0·082	—	—
			0·006	*0·007*	*0·016*	*0·020*	*0·029*	*0·038*	*0·048*	*0·056*	*0·044*	—	—
		1891	0·009	0·012	0·013	0·022	0·041	0·051	0·055	—	—	—	—
			0·007	*0·009*	*0·014*	*0·016*	*0·028*	*0·031*	*0·034*	—	—	—	—
		1901	0·009	0·011	0·016	0·020	0·025	—	—	—	—	—	—
			0·007 •	*0·009*	*0·013*	*0·014*	*0·015*	—	—	—	—	—	—
21	Cancer of Jaw	1871	—	—	0·016	0·033	0·053	0·075	0·081	0·101	0·102	0·090	0·059
			—	—	*0·009*	*0·011*	*0·017*	*0·026*	*0·035*	*0·038*	*0·027*	*0·029*	*0·018*
		1881	0·003	0·007	0·008	0·019	0·028	0·041	0·040	0·041	0·031	—	—
			0·002	*0·005*	*0·005*	*0·009*	*0·015*	*0·016*	*0·011*	*0·011*	*0·013*	—	—
		1891	0·002	0·004	0·007	0·011	0·012	0·012	0·008	—	—	—	—
			0·002	*0·003*	*0·004*	*0·007*	*0·007*	*0·007*	*0·006*	—	—	—	—
		1901	0·002	0·002	0·002	0·004	0·004	—	—	—	—	—	—
			0·002 •	*0·002*	*0·002*	*0·002*	*0·003*	—	—	—	—	—	—
22	Cancer of Breast (C)	1871	—	—	0·001	0·001	0·004	0·006	0·012	0·013	0·023	0·027	0·029
			—	—	*0·259*	*0·457*	*0·601*	*0·778*	*0·988*	*1·103*	*1·271*	*1·745*	*2·101*
		1881	0·000	0·000	0·001	0·002	0·005	0·005	0·008	0·016	0·019	—	—
			0·040	*0·127*	*0·273*	*0·460*	*0·652*	*0·802*	*0·926*	*1·064*	*1·275*	—	—
		1891	0·000	0·000	0·001	0·003	0·006	0·007	0·011	—	—	—	—
			0·041	*0·129*	*0·275*	*0·460*	*0·620*	*0·745*	*0·870*	—	—	—	—
		1901	0·000	0·000	0·001	0·002	0·004	—	—	—	—	—	—
			0·048	*0·131*	*0·269*	*0·463*	*0·580*	—	—	—	—	—	—
23	Cancer of Ovaries and Fallopian Tubes	1871	—	—	*0·046*	*0·065*	*0·125*	*0·161*	*0·210*	*0·258*	*0·270*	*0·293*	*0·296*
		1881	*0·012*	*0·021*	*0·050*	*0·109*	*0·166*	*0·221*	*0·254*	*0·296*	*0·311*	—	—
		1891	*0·015*	*0·033*	*0·072*	*0·133*	*0·201*	*0·266*	*0·291*	—	—	—	—
		1901	*0·018* •	*0·043*	*0·086*	*0·158*	*0·248*	—	—	—	—	—	—
24	Cancer of Vulva and Vagina	1871	—	—	*0·008*	*0·017*	*0·024*	*0·040*	*0·059*	*0·087*	*0·126*	*0·138*	*0·177*
		1881	*0·002*	*0·005*	*0·007*	*0·013*	*0·023*	*0·034*	*0·052*	*0·067*	*0·099*	—	—
		1891	*0·002*	*0·005*	*0·006*	*0·011*	*0·020*	*0·022*	*0·036*	—	—	—	—
		1901	*0·001* •	*0·004*	*0·007*	*0·009*	*0·014*	—	—	—	—	—	—

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APPENDIX TABLE I (*contin.*)

Fig. No.	Cause of Death	Cohort born	Age 30–	35–	40–	45–	50–	55–	60–	65–	70–	75–	80–84
25	Cancer of Uterus (Corpus and Cervix) (C)	1871	—	—	*0·321*	*0·477*	*0·609*	*0·619*	*0·642*	*0·688*	*0·753*	*0·748*	*0·724*
		1881	*0·052*	*0·146*	*0·270*	*0·396*	*0·480*	*0·538*	*0·597*	*0·614*	*0·578*	—	—
		1891	*0·054*	*0·145*	*0·233*	*0·342*	*0·430*	*0·484*	*0·488*	—	—	—	—
		1901	*0·044* *	*0·098*	*0·170*	*0·242*	*0·303*	—	—	—	—	—	—
26	Cancer of Testes	1871	—	—	0·008	0·009	0·005	0·006	0·009	0·017	0·020	0·019	0·020
		1881	0·008	0·009	0·010	0·009	0·012	0·009	0·010	0·011	0·014	—	—
		1891	0·010	0·012	0·014	0·009	0·010	0·009	0·011	—	—	—	—
		1901	0·011 *	0·015	0·011	0·012	0·008	—	—	—	—	—	—
27	Cancer of Penis and Scrotum	1871	—	—	0·007	0·011	0·021	0·028	0·044	0·055	0·080	0·086	0·094
		1881	0·002	0·003	0·007	0·010	0·016	0·018	0·033	0·026	0·041	—	—
		1891	0·001	0·002	0·004	0·005	0·010	0·010	0·017	—	—	—	—
		1901	0·001 *	0·002	0·003	0·005	0·006	—	—	—	—	—	—
28	Cancer of Prostate (C)	1871	—	—	0·002	0·008	0·035	0·099	0·278	0·616	1·142	1·963	2·830
		1881	0·001	0·001	0·003	0·010	0·042	0·117	0·296	0·657	1·304	—	—
		1891	0·001	0·002	0·003	0·011	0·039	0·120	0·248	—	—	—	—
		1901	0·001 *	0·002	0·004	0·013	0·033	—	—	—	—	—	—
29	Cancer at All Other Sites (C)	1871	—	—	0·083	0·117	0·184	0·226	0·362	0·447	0·552	0·665	0·954
			—	—	*0·064*	*0·090*	*0·125*	*0·141*	*0·262*	*0·306*	*0·331*	*0·341*	*0·781*
		1881	0·028	0·033	0·061	0·098	0·168	0·219	0·292	0·418	0·514	—	—
			0·028	*0·029*	*0·051*	*0·070*	*0·127*	*0·155*	*0·166*	*0·260*	*0·495*	—	—
		1891	0·023	0·038	0·061	0·095	0·134	0·222	0·376	—	—	—	—
			0·021	*0·029*	*0·047*	*0·075*	*0·098*	*0·146*	*0·277*	—	—	—	—
		1901	0·021	0·044	0·053	0·100	0·171	—	—	—	—	—	—
			0·020 *	*0·022*	*0·045*	*0·087*	*0·131*	—	—	—	—	—	—
30	Benign Tumours and Tumours of Undetermined Nature	1871	—	—	0·041	0·057	0·096	0·128	0·150	0·174	0·128	0·132	0·194
			—	—	*0·096*	*0·132*	*0·147*	*0·167*	*0·147*	*0·083*	*0·122*	*0·136*	*0·155*
		1881	0·026	0·038	0·060	0·085	0·111	0·115	0·130	0·105	0·126	—	—
			0·042	*0·072*	*0·122*	*0·165*	*0·150*	*0·109*	*0·104*	*0·095*	*0·142*	—	—
		1891	0·033	0·051	0·069	0·076	0·102	0·127	0·136	—	—	—	—
			0·043	*0·077*	*0·110*	*0·121*	*0·117*	*0·097*	*0·086*	—	—	—	—
		1901	0·033	0·044	0·053	0·077	0·120	—	—	—	—	—	—
			0·046	*0·069*	*0·098*	*0·089*	*0·057*	—	—	—	—	—	—
31	Leukaemia (All Types) (C)	1871	—	—	0·012	0·017	0·020	0·031	0·042	0·079	0·074	0·142	0·187
			—	—	*0·017*	*0·014*	*0·021*	*0·025*	*0·039*	*0·057*	*0·059*	*0·078*	*0·130*
		1881	0·009	0·009	0·014	0·021	0·027	0·049	0·080	0·112	0·170	—	—
			0·008	*0·011*	*0·011*	*0·019*	*0·026*	*0·040*	*0·061*	*0·087*	*0·137*	—	—
		1891	0·009	0·012	0·019	0·027	0·041	0·075	0·132	—	—	—	—
			0·009	*0·013*	*0·016*	*0·025*	*0·034*	*0·059*	*0·098*	—	—	—	—
		1901	0·014	0·019	0·022	0·043	0·054	—	—	—	—	—	—
			0·011	*0·017*	*0·021*	*0·035*	*0·050*	—	—	—	—	—	—
32	Hodgkin's Disease (C)	1871	—	—	0·009	0·013	0·020	0·028	0·033	0·036	0·038	0·044	0·031
			—	—	*0·005*	*0·006*	*0·011*	*0·015*	*0·019*	*0·023*	*0·016*	*0·024*	*0·026*
		1881	0·007	0·011	0·013	0·016	0·027	0·030	0·040	0·042	0·047	—	—
			0·005	*0·005*	*0·005*	*0·007*	*0·012*	*0·015*	*0·020*	*0·023*	*0·025*	—	—
		1891	0·011	0·016	0·018	0·019	0·026	0·039	0·046	—	—	—	—
			0·007	*0·008*	*0·010*	*0·010*	*0·016*	*0·016*	*0·027*	—	—	—	—
		1901	0·017	0·017	0·020	0·027	0·035	—	—	—	—	—	—
			0·011	*0·013*	*0·008*	*0·013*	*0·014*	—	—	—	—	—	—

APPENDIX TABLE II

PROPORTIONATE CHANGES IN DEATH RATES AT AGES 50–54, 60–64, AND 70–74. THESE ARE THE AGES ATTAINED BY THE 1901, 1891, AND 1881 COHORTS RESPECTIVELY IN THE QUINQUENNIUM 1951–55

The death rates at each of these ages are expressed as a percentage of the death rates of the 1871 cohort at these ages.

Figure No.	Cause of Death	Cohort Born	Males Age 70–	Males Age 60–	Males Age 50–	Females Age 70–	Females Age 60–	Females Age 50–
2	All Causes	1881	106·0	98·1	98·0	*92·3*	*81·6*	*90·4*
		1891	—	99·2	88·3	—	*71·3*	*73·4*
		1901	—	—	78·6	—	—	*58·5*
–	All Neoplasms	1881	107·1	99·5	97·5	*93·7*	*92·7*	*94·7*
		1891	—	113·5	101·5	—	*85·7*	*89·8*
		1901	—	—	124·2	—	—	*81·0*
3	Cancer at All Sites	1881	106·3	99·1	96·0	*92·2*	*92·9*	*94·0*
		1891	—	112·5	100·1	—	*85·0*	*89·6*
		1901	—	—	122·4	—	—	*81·9*
4	Cancer at All Sites except Lungs	1881	92·8	87·0	87·0	*90·0*	*91·5*	*92·8*
		1891	—	75·2	74·9	—	*81·1*	*86·9*
		1901	—	—	68·4	—	—	*77·6*
5	Cancer of Lungs	1881	331·7	286·4	387·9	*166·7*	*147·5*	*211·5*
		1891	—	689·2	913·6	—	*241·5*	*361·5*
		1901	—	—	1865·1	—	—	*519·2*
6	Cancer of Stomach	1881	101·1	96·8	105·6	*85·6*	*87·1*	*91·5*
		1891	—	94·1	100·2	—	*63·7*	*74·3*
		1901	—	—	92·8	—	—	*60·2*
7	Cancer of Intestines	1881	81·5	106·3	99·5	*74·6*	*102·2*	*88·9*
		1891	—	72·1	98·5	—	*78·9*	*92·9*
		1901	—	—	79·4	—	—	*73·8*
8	Cancer of Rectum	1881	77·3	91·8	93·5	*80·4*	*93·0*	*81·5*
		1891	—	63·0	81·3	—	*77·6*	*90·4*
		1901	—	—	62·5	—	—	*74·1*
9	Cancer of Liver and Gall-Bladder	1881	71·9	72·1	59·2	*63·4*	*67·6*	*57·3*
		1891	—	54·5	47·4	—	*45·5*	*42·1*
		1901	—	—	36·2	—	—	*30·5*
10	Cancer of Pancreas	1881	133·5	108·6	142·4	*119·3*	*95·8*	*118·6*
		1891	—	141·1	147·5	—	*98·6*	*116·3*
		1901	—	—	154·2	—	—	*125·6*
11	Cancer of Tongue	1881	48·1	47·6	43·7	*89·7*	*73·9*	*100·0*
		1891	—	17·1	16·2	—	*60·9*	*112·5*
		1901	—	—	6·3	—	—	*75·0*
12	Cancer of Mouth and Tonsils	1881	89·9	44·1	43·6	*126·3*	*69·2*	*87·5*
		1891	—	27·6	16·7	—	*100·0*	*37·5*
		1901	—	—	17·9	—	—	*62·5*
13	Cancer of Pharynx	1881	90·5	65·3	87·5	*187·0*	*81·3*	*83·3*
		1891	—	51·6	56·3	—	*187·5*	*83·3*
		1901	—	—	50·0	—	—	*158·3*
14	Cancer of Larynx	1881	70·7	71·2	75·3	*75·0*	*105·4*	*111·5*
		1891	—	50·7	44·3	—	*62·2*	*103·9*
		1901	—	—	37·1	—	—	*50·0*
15	Cancer of Oesophagus	1881	76·5	56·9	54·3	*116·7*	*86·8*	*88·7*
		1891	—	40·5	31·5	—	*74·6*	*61·3*
		1901	—	—	27·4	—	—	*51·6*
16	Cancer of Kidneys and Adrenals	1881	181·5	100·0	129·6	*144·1*	*86·7*	*133·3*
		1891	—	153·4	140·7	—	*106·7*	*111·1*
		1901	—	—	200·0	—	—	*116·7*
17	Cancer of Urinary Bladder	1881	132·3	120·2	129·2	*100·6*	*139·0*	*81·8*
		1891	—	148·6	164·6	—	*108·5*	*127·3*
		1901	—	—	183·3	—	—	*113·6*

—contin.

Figure No.	Cause of Death	Cohort Born	Males Age 70–	Males Age 60–	Males Age 50–	Females Age 70–	Females Age 60–	Females Age 50–
18	Cancer of Skin (excluding Scrotum)	1881	61·6	84·0	86·7	*80·0*	*76·2*	*81·8*
		1891	—	61·3	66·7	—	*69·1*	*72·7*
		1901	—	—	66·7	—	—	*54·5*
19	Cancer of Lips	1881	41·6	52·5	83·3	*100·0*	*100·0*	*100·0*
		1891	—	22·5	33·3	—	*50·0*	*0·0*
		1901	—	—	8·3	—	—	*0·0*
20	Cancer of Bones (excluding Jaw)	1881	74·5	128·6	134·5	*67·7*	*106·7*	*103·6*
		1891	—	87·3	141·4	—	*75·6*	*100·0*
		1901	—	—	86·2	—	—	*53·6*
21	Cancer of Jaw	1881	30·4	49·4	52·8	*48·2*	*31·4*	*88·2*
		1891	—	9·9	22·6	—	*17·1*	*41·2*
		1901	—	—	7·5	—	—	*17·7*
22	Cancer of Breast	1881	82·6	66·7	125·0	*100·3*	*93·7*	*108·5*
		1891	—	91·7	150·0	—	*88·1*	*103·2*
		1901	—	—	100·0	—	—	*96·5*
23	Cancer of Ovaries and Fallopian Tubes	1881	—	—	—	*115·2*	*121·0*	*132·8*
		1891	—	—	—	—	*138·6*	*160·8*
		1901	—	—	—	—	—	*198·4*
24	Cancer of Vulva and Vagina	1881	—	—	—	*78·6*	*88·1*	*95·8*
		1891	—	—	—	—	*61·0*	*83·3*
		1901	—	—	—	—	—	*58·3*
25	Cancer of Uterus (Corpus and Cervix)	1881	—	—	—	*76·8*	*93·0*	*78·8*
		1891	—	—	—	—	*76·0*	*70·6*
		1901	—	—	—	—	—	*49·8*
26	Cancer of Testes	1881	70·0	111·1	240·0	—	—	—
		1891	—	122·2	200·0	—	—	—
		1901	—	—	160·0	—	—	—
27	Cancer of Penis and Scrotum	1881	51·3	75·0	76·2	—	—	—
		1891	—	38·6	47·6	—	—	—
		1901	—	—	28·6	—	—	—
28	Cancer of Prostate	1881	114·2	106·5	120·0	—	—	—
		1891	—	89·2	111·4	—	—	—
		1901	—	—	94·3	—	—	—
29	Cancer of All Other Sites	1881	92·3	77·6	91·3	*66·9*	*63·4*	*132·8*
		1891	—	100·8	72·8	—	*105·7*	*78·4*
		1901	—	—	92·9	—	—	*104·8*
30	Benign Tumours and Tumours of Undetermined Nature	1881	98·4	86·7	115·6	*116·4*	*70·8*	*102·0*
		1891	—	90·7	106·3	—	*58·5*	*79·6*
		1901	—	—	125·0	—	—	*38·8*
31	Leukaemia (All Types)	1881	135·1	190·5	135·0	*232·2*	*156·4*	*123·8*
		1891	—	314·3	205·0	—	*251·3*	*161·9*
		1901	—	—	270·0	—	—	*238·1*
32	Hodgkin's Disease	1881	123·7	121·2	135·0	*156·3*	*105·3*	*109·1*
		1891	—	118·2	130·0	—	*142·1*	*145·5*
		1901	—	—	175·0	—	—	*127·3*

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GENERAL PRACTICE ON A NEW HOUSING ESTATE

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In recent years there has been an increasing recognition of the value that doctors' records can have in throwing light both on the working of general practice and on morbidity in the population. The present study consists of an analysis of the records kept by the general practitioners working on a new housing estate and forms part of an investigation into the health of a rehoused population.

Materials and Methods

The estate is situated about 15 miles from the centre of London and was built by the London County Council between the years 1948 and 1952 as part of the post-war housing programme. It comprises just over 4,000 houses, with the necessary shops, schools, etc. Almost all the families on the estate came from Greater London. About two-thirds of them were rehoused from the Council's waiting list, principally on the grounds of overcrowding or ill-health. The remainder transferred to the estate at their own request from other L.C.C. accommodation which they were occupying in the London area. The total population of about seventeen thousand consists mainly of couples with children, the average size of the families being four persons.

There are six doctors practising on the estate. Five of these form a partnership, two members of which also have surgeries off the estate. About 13,000 people are registered with this partnership, which is conducted from two surgeries on the estate, each having two consulting rooms and a common waiting-room. The sixth doctor has about 3,000 patients on his list and practises from a house which he occupies on the estate.

For the purpose of this study, all six doctors adopted a simple system of record keeping, using a modified form of the National Health Service card. In respect of each consultation, the doctor recorded on the patient's card the date, place, diagnosis or other reason for attendance, treatment, certificates issued, and details of any referral.

The recording system was brought into use about 2 months before the beginning of the year 1953, in order to allow the practitioners to familiarize themselves with the procedure. No difficulties arose in recording consultations made in the surgeries, but it is possible that the reporting of home visits was somewhat less reliable. Four of the doctors recorded home visits on slips from which the entries were afterwards transcribed on to the patients' cards by a secretary. The other two doctors preferred to take the patients' cards with them and complete them at the time of the consultations. On some occasions, however, the visits were made in response to a telephone message or note, asking the doctor to call but without giving the patient's name. Where this occurred, the doctor recorded the consultation and entered it on the card after his return to the surgery. In the course of the day-to-day working of busy practices, it is possible that certain errors were introduced but they are likely to have been small.

The Sample

Almost all the patients on the doctors' lists were resident on the estate and those who were not residents (224 registered with the partnership, and 12 with the other doctor) have been omitted from this study.

A 25 per cent. sample of the addresses on the estate was drawn using a set of random numbers, and the

analysis was carried out on the records of the registered patients who lived at those addresses during the year 1953. The information on the record cards was then coded, transferred to punch cards, and sorted mechanically.

There were 1,018 addresses in the sample. Owing to removals, 77 of these houses had two families living in them during the year, making a total of 1,095 families comprising 4,749 persons. These families ranged in size from one to eleven, with an average of 4·3 persons. About half of the families consisted of either four or five persons. In nearly three-quarters of the families in the sample every member was registered with a doctor on the estate for at least some part of the year. A further 17 per cent. of the families had some, but not all, members registered, and 9 per cent. had no-one registered with a doctor on the estate.

Thus, of the 4,749 persons in the sample addresses, 4,067 (86 per cent.) were registered with the doctors on the estate. About 18 per cent. of the total registered population were on the doctors' lists for less than 12 months of the year, the average duration for all patients being 10·9 months. 80 per cent. of the patients were registered with the partnership, and the remainder with the doctor who practised alone. Only ten patients in the sample consulted with both practices.

As would be expected in a community of this kind, the size of the practices fluctuated very little throughout the year; the average registered population in the sample, based on the actual numbers at the end of each quarter, was 3,710.

Results

Description of the Registered Population.—Table I shows that the distribution of age groups in the practice population differed appreciably from that of Greater London and from that of England and Wales as a whole. The principal characteristics of this rehoused population were the high proportion of young children and the relatively small number of old persons. These features, which are common to all new housing estates, are likely to affect the nature of the sickness occurring among the population.

Table I
AGE DISTRIBUTION OF REGISTERED POPULATION

Age Group (yrs)	Registered Population (per cent.)	Greater London (per cent.)	England and Wales (per cent.)
0–4	13·2	8·2	8·5
5–14	25·1	12·5	13·7
15–44	43·2	44·0	42·8
45–64	9·0	24·6	24·1
65 and Over	3·1	10·7	10·9
No Record except 21 and over	3·7	—	—
No Records at all ..	2·7	—	—
Total	100·0	100·0	100·0

Table II shows the proportion of females among the registered patients, in Greater London, and in England and Wales. There are slight differences in certain age groups.

Table II
PROPORTION OF FEMALES

Age Group (yrs)	Registered Population (Per cent. Female)	Greater London (Per cent. Female)	England and Wales, 1951 (Per cent. Female)
0–4	50·6	50·2	49·0
5–14	50·8	49·0	49·0
15–44	53·2	52·6	50·6
45–64	49·2	54·2	53·7
65 and Over	57·1	61·2	58·5
All Ages	51·8	53·2	52·0

Table III shows the social class distribution of the principal wage-earners of the 998 families to which the registered patients belonged, and that of occupied males in Greater London and England and Wales. Although differences in age do not permit a strict comparison, certain features of the social class composition of the estate population are worth noting. Families in Social Class III predominate and the proportions in Social Classes I and II are much smaller, and that in Social Class V a little smaller than in Greater London and England and Wales.

Table III
SOCIAL CLASS DISTRIBUTION

Social Class	Registered Population* (per cent.)	Greater London† (per cent.)	England and Wales† (per cent.)
I, II	6·3	21·4	19·1
III	64·4	54·7	51·4
IV	10·8	10·7	16·3
V	8·2	13·2	13·2
Retired, Disabled, and No Record	10·3	—	—
Total	100·0	100·0	100·0

* Principal wage-earners of patients' families.
† Males aged 15 and over.

Half of the principal wage-earners were engaged on skilled manual work, and about one-fifth in non-manual work, of whom about one-third were clerical workers.

Just over one-third of the total registered population was gainfully employed, and 30 per cent. of these were working in engineering trades, metal manufacture, transport, or communications. About 40 per cent. of the total patients were schoolchildren or pre-schoolchildren, and a further 18 per cent. were housewives who did not go out to work.

Consultations.—In this investigation, a "consultation" included not only a meeting between the

patient and the doctor at the surgery or in the home, but also treatment or advice given in response to a message received by note or telephone.

During the year, 76 per cent. of the registered population in the sample consulted the doctor at least once. The consultation rate is a unit of measurement that has been commonly employed in studies of general practice to assess the extent to which the service is utilized. There is, however, at the present time, no generally accepted base for the calculation of rates of this kind. Accordingly, in this paper, the rates will be expressed as the average number of consultations per patient per annum based on:

(*i*) the total practice population;
(*ii*) the average number of patients on the lists, calculated from the actual numbers registered at the end of each quarter;
(*iii*) the number of patients who consulted during the year.

The total number of consultations among the 3,081 patients who consulted was 15,286. This gives consultation rates for the sample population of 3·8 per person registered, 4·1 per person based on the average registered population, and 5 per person consulting. These figures may be compared with 5 consultations per person registered found in the studies of Bradford Hill (1951) and McGregor (1950), 5 per person based on the average registered population and 7 per person consulting reported by Backett, Heady, and Evans (1952), and 3·3 per person based on a mid-year registered population found by Fry (1952). Logan (1953), reporting on eight practices, found a range of from 2·9 to 4·7 consultations per person based on the average registered population.

Since 45 per cent. of the families on the estate had been granted some priority for rehousing on medical grounds, it might have been expected that their consultation rates would have exceeded those in other areas. That this was not so is partly due to the low mean age of the population.

Fig. 1 shows the consultation rates per 100 average registered population by sex and age group.

Although the older patients consulted the doctor more often than the young ones, the fact that old people formed only a small proportion of the population meant that their demands on the doctors accounted for a much smaller fraction of the consultations that took place. Patients under 15 accounted for 36 per cent., and those between 15 and 44 for 45 per cent. of all consultations. Thus four out of five consultations were made by people under

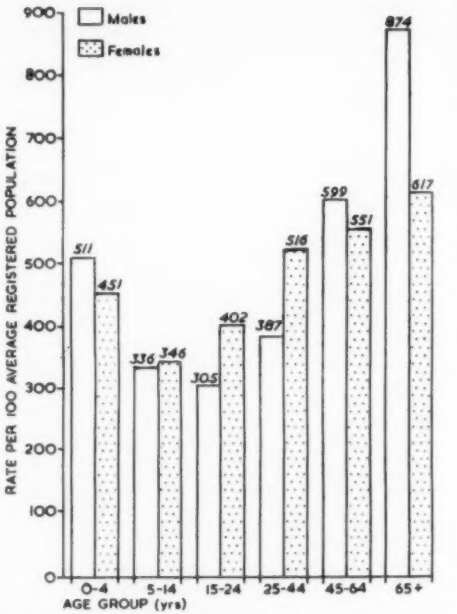

FIG. 1.—Consultation rate per hundred persons registered by sex and age group.

45 years of age. The patients aged 65 and over, *i.e.*, those whose consultation rates were by far the greatest, made little more than 5 per cent. of the total consultations. Women made more consultations than men (54 and 46 per cent. respectively) and more than one-quarter of all consultations were made by women aged 15 to 44.

About one-quarter of the registered patients did not consult the doctor at all during the year (males 27 per cent. and females 22 per cent.). A further 38 per cent. who did consult had less than four consultations (Fig. 2, opposite).

In each of the four age groups, 0–14, 15–44, 45–64, and 65 and over, about 16 per cent. of patients accounted for half the consultations. Nearly 7 per cent. of all registered patients consulted the doctor on twelve or more occasions, and accounted for 30 per cent. of the total. This state of affairs has been reported in other studies of general practice.

When the registered patients were classified according to the social class of the principal wage-earner of each family, a slight social gradient appeared in the use of the general practitioner services. The average number of consultations per person increased from 3·2 for Social Classes I and II

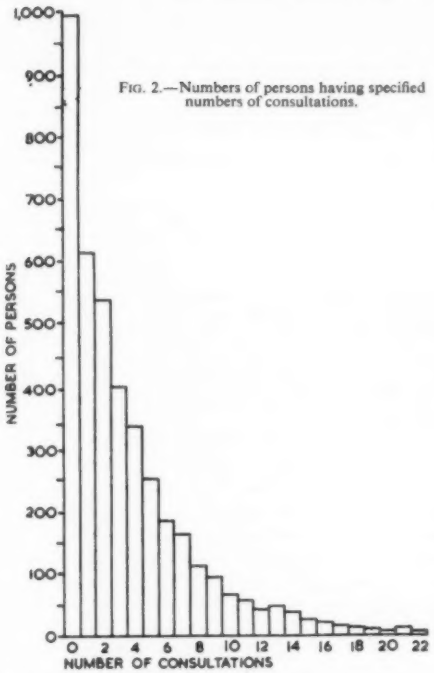

FIG. 2.—Numbers of persons having specified numbers of consultations.

to 3·8 for Social Class V (Table IV). These differences are statistically significant, but 6 per cent. of the registered patients were not assigned to a social class. These were patients from families dependent on pensions and allowances and those for whom the relevant information was not obtained. Since this group included most of the elderly patients, the consultation rate was higher than that for the classified groups.

TABLE IV

CONSULTATION RATES AND ILLNESS RATES BY SOCIAL CLASS

Social Class ..	I and II	III		IV	V	No Record
		c*	a,b,d,e			
Consultation Rate ..	3·2	3·3	3·7	3·7	3·8	4·9
Illness Rate ..	2·1	2·0	2·4	2·3	2·4	2·6

* c = clerical

Seventy consultations (0·46 per cent.) took place between 8 *p.m.* and 8 *a.m.*, which is considerably less than the 0·79 per cent. reported by Backett, Heady, and Evans (1952) and the 0·75 per cent. reported by Fry (1952).

EPISODES OF ILLNESS.—The definition of an episode of illness implies some reference to time, but the measurement of duration of sickness is fraught with difficulty. In most instances neither onset nor termination is clearly defined. On the other hand, the period during which the patient is under medical care can be established, and can be used both to define an episode of illness and to measure its duration. It can also be used to find the number of episodes of illness in a population, and to compare the effects of different diseases and the behaviour of different age groups.

Accordingly, in this study, the duration of an episode of sickness has been defined as the period between the first and last consultations for that episode, including consultations for debility following the illness. Where a person was suffering from more than one disease at the same time, the duration of each episode was measured in the same way, treating the conditions separately. If there was only one consultation, the episode was regarded as lasting for one day. In chronic conditions, an episode was regarded as continuing so long as the patient was under care. For chronic diseases not needing continuous supervision, exacerbation of illness was considered to mark the onset of a new episode.

During the year there were 9,479 episodes of illness among 3,043 patients: 2·3 per person registered, 2·6 per person based on the average registered population, and 3·1 per person consulting.

The distribution of the number of episodes of illness among the total registered population is shown in Fig. 3. One-quarter had no illness, about one-half had from one to three illnesses, and the

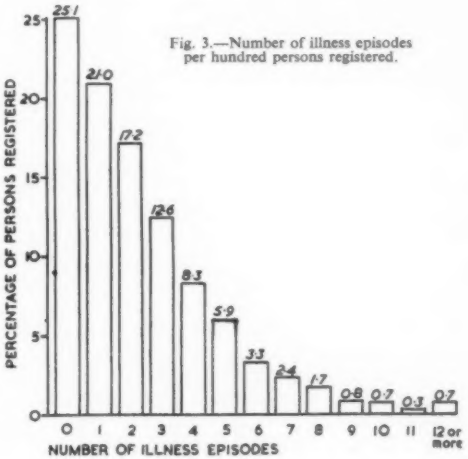

Fig. 3.—Number of illness episodes per hundred persons registered.

remaining quarter had four or more illnesses during the year. About 5 per cent. of the registered population had more than seven illnesses in the 12 months.

Fig. 4 shows the illness rates (per 100 average registered population) by sex and age group. Among males the rate fell from infancy to adulthood and then increased with age. For females, the rate fell from infancy to adulthood, rose steeply for the next 20 years, and remained relatively steady thereafter.

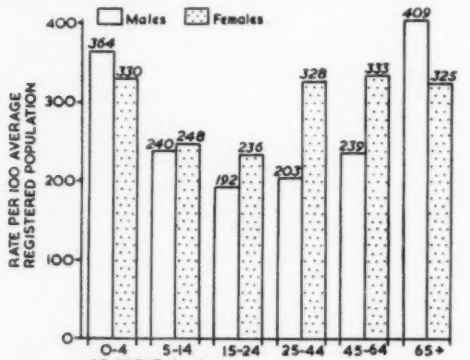

Fig. 4.—Illness episodes per hundred persons registered by sex and age group

In infancy and old age illness rates were higher for males than for females; at all other ages the female rates were greater. Figs 1 and 4, showing the consultation and illness rates, show that the young adult males (15–44) were the most healthy and the men aged 65 and over the least healthy. This is not in accord with the findings of other workers. The records of the housing authority showed that 34 per cent. of the men aged 65 and over had been granted priority for rehousing on medical grounds, compared with only 18 per cent. of the women. The incidence of chronic conditions and defects recorded at the first consultation was 57 and 30 per cent. for elderly men and elderly women respectively.

In the age group 45–64, men had fewer illnesses but more consultations than women. The higher consultation rate for men was largely due to the fact that most of them had to obtain National Insurance certificates during illness. For this age group, 23 National Insurance certificates were issued for every 100 males registered, compared with 5 certificates for every 100 females (1 certificate for every 2·4 consultations for men, as against 1 for every 10 consultations for women). This suggests that the requirements of the present system of National Insurance benefits tend to increase the number of consultations made by wage-earning patients.

Among patients who consulted during the year, about 80 per cent. had an average of from one to three consultations for each episode of sickness; 3 per cent. had five or more.

The number of consultations per illness appeared to increase with age. For males it rose from an average of 1·4 consultations per illness at age 0–4, to 2·5 at age 45–64, and fell to 2·1 for 65 and over. For females the average was 1·4 consultations per illness at ages 0–4 and increased to 1·9 at 65 and over.

The number of consultations per illness fell slightly as the number of illnesses increased (1·9 for patients having one illness, 1·5 for patients having five illnesses, and 1·3 for patients having twelve or more). This trend was probably influenced by the fact that multiple conditions could often be treated together at the same consultation.

The social gradient was less marked for illnesses than for consultations (Table IV).

Duration of Sickness.—70 per cent. of all episodes of illness were dealt with by single consultations and were thus regarded as being under care for one day. Nearly one-quarter of the episodes lasted for from 2 to 30 days, 7 per cent. for more than 30 days and only 3 per cent. for more than 90 days.

By adding together the number of days that each patient was under care, it is possible to obtain an assessment of the total period of sickness for each person during the year. Although this is an exceedingly rough measure, it does allow certain comparisons to be made.

The distribution of total days' sickness among the registered population is shown in Table V. More than half the patients were under care for less than 5 days, one-quarter for from 5 to 30 days, and only about 8 per cent. for more than 90 days.

Table V
DAYS OF SICKNESS, UNDER CARE, DURING 1953

Days of Sickness under Care	Percentage of Registered Population
0	25·1
1–4	32·6
5–14	15·1
15–30	9·7
31–90	9·8
More than 90	7·7

If the number of days' sickness is compared by sex and age group, the outstanding feature among males is a steady increase with age in those having more than 30 days' sickness (from 11 per cent. in males under 15, to 47 per cent. at 65 and over). Among females the rate increased from 10 per cent.

for those under 15 to 33 per cent. for those aged 45–64, and then fell slightly to 30 per cent. for those aged 65 and over.

CERTIFICATES.—About one certificate was issued for every patient consulting and one certificate for every five consultations. About two-thirds of these certificates were required by the National Insurance regulations.

PRESCRIPTIONS.—The prescription rate may be regarded as one measure of the treatment received by the patient. In this study each item of treatment prescribed was regarded as a single prescription. 15,098 prescriptions were issued to 2,898 patients in the sample, 99 prescriptions for every 100 consultations. The prescription rates were 3·7 per person registered, 4·1 per person based on the average registered population, and 4·9 per patient consulting. These rates approximate closely to the consultation rates and are almost identical with those reported by Backett, Heady, and Evans (1954).

Only about 5 per cent. of the registered population consulted the doctor without obtaining prescriptions and most of these consultations were for reasons other than sickness. Nearly one-quarter of the patients had one or two prescriptions and accounted for about 10 per cent. of all the prescriptions issued.

10 per cent. of patients had ten or more prescriptions, and accounted for 38 per cent. of the total. A small group (1·5 per cent.) had twenty or more prescriptions (11 per cent. of the total number issued).

56 per cent. of the prescriptions were issued to females. The trends with age in the prescription rates for the two sexes (shown in Table VII, below) are similar to those in the consultation rates, except that the male patients aged 45–64 had fewer prescriptions but more consultations than the females. The young adult males (15–24 years) had the fewest prescriptions, whereas the men aged 65 and over had proportionately more than any other group.

REFERRALS.—During the year there were 31 referrals for every 100 patients registered. This figure approximates to the 35 per 100 reported by Backett, Heady, and Evans (1954). In the eight practices studied by Logan (1953) the referral rates per 100 practice populations ranged from 6·4 to 33 (average about 16).

Table VI shows that 70 per cent. of all referrals were to hospitals as out-patients and 10 per cent. as in-patients.*

* An in-patient referral relates to a person sent as an inpatient by the doctor but not necessarily accepted as such by the hospital. It is a statement of the doctor's intention rather than of what may actually have occurred.

TABLE VI
REFERRALS

Type of Referral		Hospital		"Other"	Total
		In-patient	Out-patient		
Referrals	No.	113	809	240	1,162
	Per cent.	10	70	20	100
Persons	No.	109	658	223	909
	Per cent.	12	72	26	

About a quarter of the patients who consulted were sent to hospital, and four out of every five of these went as out-patients. Nearly one in five of the people who were sent as out-patients were referred more than once, although not necessarily for the same illness.

Rather more women then men were sent to hospital, 57 per cent. of the people referred as in-patients and 54 per cent. of the out-patients being females.

Hospital referral rates were 18·3 per 100 patients registered, 20 per 100 based on the average registered population, and 24 per 100 patients consulting.

Fig. 5 shows referral rates to hospitals by age and sex based on the average registered population. For males the rates rose steadily with age; for females they increased to ages 45–64, and then fell.

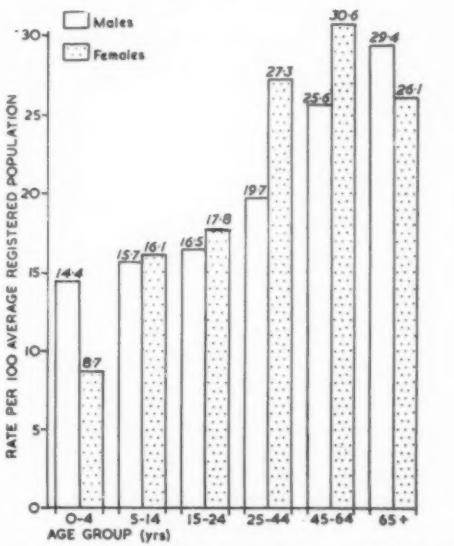

FIG. 5.—Referrals to hospital per one hundred persons registered by sex and age group.

One-fifth of the referrals were to agencies other than hospitals; 34 per cent. of these "other" referrals were to ophthalmologists and opticians, 28 per cent. to district nurses, and 17 per cent. to dental surgeons. The remainder included referrals to hospital for blood counts, or to health visitors, and specimens sent to hospital laboratories for diagnostic tests.

Discussion

The population of this housing estate had certain characteristics which would be expected to influence the incidence of sickness and hence the extent to which the services of the general practitioners would be utilized. A considerable proportion of the families had been rehoused on grounds of ill-health, generally of a chronic nature, a factor which would tend to increase the usage of the local medical services. On the other hand, the age composition of the estate population, with its high proportion of school-children and relatively small number of old people, would be likely to reduce the extent of sickness below that of a more representative sample of the general population. The application to the doctors' records of four yardsticks (the rates for consultation, illness, prescription, and hospital referral) suggests that these two influences have brought about a degree of usage of the general practitioner services which differs little from that found in other areas.

Indeed, a significant feature of the results of this inquiry is the considerable agreement with the findings of other workers in this field, notably those of Fry (1952), Logan (1953), and Backett, Heady, and Evans (1954). In all these studies, about three-quarters of the registered patients consulted in the course of 12 months, an average of from three to five consultations per person. There was more work among women than men, and more work among the old than the young. The healthiest group comprised the adolescent and young adult males.

In the estate population, the highest rates for consultations, illnesses, prescriptions, and hospital referrals occurred among the men aged 65 and over (Table VII). Fry (1952), Logan (1953), and Backett, Heady, and Evans (1954) all reported higher consultation rates among the women of this age group. It is probable that the rehousing process entails some factor of selection which would account for this difference. Most women of this age still have housekeeping responsibilities. If unfit, an elderly housewife would be unlikely to seek to be rehoused on an outlying estate, but this would not necessarily be true of an elderly unfit husband. Support for this view is provided by the observation that more elderly men than elderly women had medical priority for rehousing and that the incidence of chronic medical conditions and defects recorded at the patients' first consultations was higher for males of this age than for females. The small number of old people living on this estate are evidently unrepresentative of their age group.

Table VII

CONSULTATION, ILLNESS, PRESCRIPTION, AND HOSPITAL REFERRAL RATES PER PERSON, BASED ON THE AVERAGE REGISTERED POPULATION

Age-Group (yrs)	Sex	Consultation Rate	Illness Rate	Prescription Rate	Hospital Referral Rate
0–4	Male	5·1	3·6	5·0	0·14
	Female	4·5	3·3	4·6	0·09
5–14	Male	3·4	2·4	3·3	0·16
	Female	3·5	2·5	3·4	0·16
15–24	Male	3·1	1·9	2·7	0·17
	Female	4·0	2·4	3·2	0·18
25–44	Male	3·9	2·0	3·1	0·20
	Female	5·2	3·3	4·9	0·27
45–64	Male	6·0	2·4	5·0	0·26
	Female	5·5	3·3	5·7	0·30
65 and Over	Male	8·7	4·1	8·6	0·30
	Female	6·2	3·3	7·1	0·26

The concentration of work among a relatively small proportion of the patients is another common feature of general practice. In this study, as in that of Backett, Heady, and Evans (1954), 16 per cent. of the registered patients accounted for one-half of the consultations. The 7 per cent. of patients who took up 30 per cent. of the consultations represent the "hard core" of the doctors' work. Further attention will be given to this group in a later stage of this investigation.

Examination of the period under care is a tentative attempt to introduce measurement into an important but difficult aspect of morbidity studies. It has been of value here in revealing the large proportion of illnesses which doctors saw only once and the small proportion treated over a long period. It showed that more than half of the registered patients were under care for less than 5 days in the year, and the extent to which duration of sickness is a function of age. Further use will be made of this measure in the succeeding stages of the analysis, which will be concerned with the nature of the sickness occurring in the sample population.

In studies of this kind there is need for agreement on the way in which the information is to be presented. This is particularly true in regard to such matters as the calculation of consultation rates. Hitherto, a variety of denominators has been employed, including the total practice population, the number of patients consulting, and the numbers at risk at the mid-year. In this paper use has been made of the average registered population calculated from the actual numbers of patients on the lists at the end of each quarter. While each of these denominators has certain advantages, it is also desirable that there should be a generally accepted basis for the determination of rates.

The task of keeping the records was not found to be unduly onerous by the practitioners concerned and was carried out with the minimum of clerical help. In the course of time, however, the records were found to be of assistance in providing an up-to-date medical history of the patients. They were particularly helpful to the members of the partnership, where at least half of the patients consulting were seen by more than one doctor, and 14 per cent. saw three or more doctors in the year. This is likely to be a characteristic that is common to partnership practice of this kind, and it is one in which a simple system of record keeping can be especially useful.

The value of such a system to the doctors themselves and for research purposes, which has been noted by others, has been confirmed by this study.

Summary

An analysis has been carried out of the records kept for the year 1953 by six doctors practising on a new housing estate near London. The population of the estate is described in terms of age, sex, and social class.

76 per cent. of the registered patients consulted a doctor at some time during the year, the consultation rate per person being 4·1 based on the average registered population; females had more consultations than males; 80 per cent. of all consultations were made by people under the age of 45.

One-sixth of the patients accounted for about half of the consultations, and 30 per cent. of the consultations were made by the 7 per cent. of registered patients who consulted twelve or more times in the year. The consultation rates showed a slight, but statistically significant, social gradient, with more consultations among patients in the lower social classes.

The illness rate was 2·6 per person, and was higher among males in infancy and old age, and among females between the ages of 5 and 64.

A method of estimating the duration of sickness in terms of the period under medical care was employed. This showed that 70 per cent. of all illnesses were dealt with in single consultations, and that only 3 per cent. of illnesses were under care for more than 90 days. Over half of the practice population were under care for less than 5 days, and only 8 per cent. for more than 90 days.

The proportion of patients having more than 30 days sickness generally increased with age, but there was a slight fall among the women aged 65 and over.

Certificates were issued at the rate of about one for every five consultations, two-thirds of these being necessitated by the requirements of the National Insurance regulations.

Prescriptions were issued at the rate of about one per consultation, the prescription rate being 4·1 per person. Only 5 per cent. of the practice population consulted the doctor but did not obtain a prescription.

About 30 per cent. of the patients who consulted a doctor were referred outside the practices, 80 per cent. of all referrals being to hospitals, either as in-patients or as out-patients. The person-hospital referral rate was 20 per 100, and there were 31 referrals for every 100 registered patients.

Males over 65 had the highest rates for consultations, illnesses, prescriptions, and referrals. The reason for this is discussed.

The value of record-keeping by general practitioners is stressed, together with the need for a generally accepted method of expressing rates in studies of this kind.

We should like to thank Professor J. M. Mackintosh for his constant support and encouragement, and Miss Jane Cooper and her assistants whose management of the records has made this study possible.

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